BRAIN.

PARTS III. & IV., 1892.

Original Articles.

ON THE FUNCTIONAL ATTRIBUTES OF THE CEREBRAL CORTEX

BY AUGUSTUS D. WALLER, M.D., F.R.S.

SUMMARY OF CONTENTS.

Part I. On the psychological interpretation of some physiological data.

§ 1. INTRODUCTION.—May we rest satisfied with the description of "motor" and "sensory" centres in the brain? The terms are frequently used in a vague and misleading manner, all the more so that they appear to be precise. We are apt to think we know what we are talking about in using such apparently specific designations as "motor centres" and "sensory centres." But cortical functions cannot be described in terms of pure motor and sensory events.

The original standpoint of Hitzig and of Munk. The motor area according to Hitzig is a muscular sense area, according to Munk a general sensory area. Quotations from Hitzig and from Munk.

§ 2. SENSORY AND MOTOR CENTRES.—Analysis of the terms motor and sensory as applied to nerve centres and to neural processes. As regards the highest centres we have very little ground for admitting any distinction between motor and sensory. One and the same central process is the cause of the motor impulse and of the sensation. A centre does not feel; it is the seat of a process that causes sensation.

§ 3. CORTICAL CENTRES. — The antithesis between the popular terms "motor" and "sensory" has led to the "carrot and turnip" theory of cortical centres. The cortical neural process is senso-motor.

Similarity of the ideas expressed in dissimilar words by Hitzig, Munk and Bastian. The two sides of the shield viewed by Ferrier and by Bastian. Bastian's repudiation of the motor function of the Rolandic area. His kinæsthesis diagram; the pyramidal tract not efferent, but internuntial. James' comparison of the Rolandic area with the mouth of a funnel. Significance of experiments by Bubnoff and Heidenhain.

Part II. On the physiological significance of some psychological data.

§ 4. Elementary Considerations. Neural gradation. The Jacksonian scheme. The simplest sensation is a relation between two factors. The four R's. Neural synthesis. Similarity of view in Wundt's theory of an apperceptive centre, Jackson's highest level centre, Ferrier's doctrine *re* pre-frontal cortex, Munk's psychical area. The unity of consciousness and the localisation of cortical function. Halo symptoms of organic lesions.

§ 5. The Sense of Effort.—Sensations in association with molecular processes taking place in senso-motor nerve centres. Attention is increased senso-motivity. Contrast. The initiation of action in a highest centre is demonstrably attended with a consumption of force-producing material. The sensations of "effort" and of "fatigue" depend upon changes originating at the periphery and in the centre. So-called demonstration of the lack of sensibility accompanying the action of so-called motor centres. The direct responsibilty of muscle.

§ 6. Apperception.—It is a misrepresentation of Wundt's position to make him responsible for an animistic *faculty* of apperception.

§ 1—Introduction.

The following pages[1] embody an attempt in some degree to estimate the functional significance of the experimental data acquired during the last two decades on the cerebral cortex.

The immediate occasion of the attempt was a study of the "sense of effort" which has occupied my thoughts at intervals during the last few years, and has inevitably connected itself with reflections upon the psychophysiological

[1] This paper is in the same grooves of thought as a paper published in Brain last year on the Sense of Effort.

It contains in fact most of the considerations that form an area of indistinct consciousness surrounding the area of more distinct consciousness relating to the Sense of Muscular Effort.

The two affirmative propositions—"This is a fact," "I think this is a fact"—bear widely different significations in common language, but are psychologically of identical meaning; they give expression to an identical psychological conclusion, and in scientific analysis as distinguished from special pleading the first assertion, however clearly and dogmatically it be propounded, can never carry more weight than the second; conviction in the mind of an onlooker or listener should depend, not upon the sense of certainty felt to be possessed by the utterer of a conclusion, but upon the verifiable premises of that conclusion exposed to critical examination.

I have attempted to make some distinction between the more psychological and the more physiological aspects of the question; a satisfactory distinction has been impossible, physiological descriptions necessarily

interpretations that have been given of cortical activity, more especially as presented in the writings of Hitzig, Ferrier, Munk and Bastian. It has no less inevitably connected itself with purely psychological considerations, as presented more especially in the writings of Bain, Wundt and James.

These are indeed by no means a complete list of the considerations and data underlying the particular topic of study designated by the title "sense of effort"; the significance of the phenomena of attention, and more especially their relation to "motor" and "sensory" events continually presented itself; the phenomenal content, common to the psychological terms—"will," "memory," "effort," "attention," had to be reckoned with; the everlasting and never solved problems coming to our minds under the various verbal labels "free will," "determinism," "predestination," "fatalism," "moral responsibility," "automatism," &c., occasionally obtruded themselves.

I allude to these ultra-scientific matters, only to say that, in my opinion, the superficial logical connection between the study of neural phenomena, and an "*explanation*" of the final causes of neural processes, by animistic references to "force," "matter," "will," "faculties," "mind," in short by any kind of disguised anthropomorphic materialism, is in relation to positive knowledge neither scientific nor ultra-scientific but anti-scientific. Beyond every "explanation" we are faced by the insoluble mystery, confessed but not

include psychological inference, psychological descriptions are in part based upon physiological phenomena, seen, reported or imagined. The attempt has, however, led me to separately present in § 4 a least positive group of considerations which, although I have not been able to omit them altogether, can be omitted in the reading without prejudice to the main argument. In common with many thinkers, I regard logical forms of verbalisation as an outward and visible sign of the inward processes of ratiocination which are functional attributes of the cerebral cortex; this belief has to a considerable extent governed my mode of thought, and I could not suppress § 4 without committing a reticence of exposition. I am as sensible as the most exacting critic can be of the unsubstantial and uncertain character of any inferences from external formulations of thought, to the hypothetical neural processes of which sensation, perception and ratiocination are the subjective concomitants.

explained in the words:—"It is the spirit that quickeneth," recognised but not comprehended in Aristotle's conception of the immaterial Psyche—nutritive, sentient and intelligent.

Therefore, in spite of its superficial logical consequence, I shall premise, as a fundamental postulate, that voluntary movement, objectively studied, is reflex movement. It is no addition to description, and, although at times convenient or even necessary, in many instances it serves to arrest description, to refer voluntary movement to Will. What is Will? What is Force? What is Matter? We do not know. Our business as students of nature is with the relations between phenomena—subjective phenomena in our own consciousness mentally projected outside our own consciousness, and as a general working hypothesis, believed to be the effects of corresponding objective phenomena around us. What is consciousness? We do not know.[1]

But assuming the real existence of ourselves and of our surroundings, we may, having recognised that we do not actually *comprehend* the verbo-mental images—Force, Matter, Motion, investigate and describe the phenomenal relations which, for convenience, we attribute to these elementary notions. Description comes to its dead end when we have traced back the phenomenal sequence to that point where we find it necessary to evoke an imaginary substantive force. A neural process for instance ceases to be described when it is traced back to the force of "will," or to the property of "excitability." The fault of which we are most often guilty, even when we have fully recognised the distinction between comprehensible relation and incomprehensible essence, is to *prematurely* invoke a final cause. The fault of which we are sometimes guilty is, after having invoked a final cause, *a quo* or *ad quem*, whether prematurely or not, to proceed through the dead end as if the phenomenal sequence were unbroken. And still grosser faults are possible—faults of observation, faults of interpretation, faults of language, and faults of candour.

[1] What consciousness is we do not know. All that we do know is consciousness. Obviously the paradox depends upon thimble-rigging with the word "know."

The particular theme now submitted to analysis is the functional significance of the cerebral cortex; the question to be answered may be put thus: Have we reached any true dead end to knowledge in the conclusion that the cerebral cortex contains "sensory centres" and "motor centres," and if so, what signification do we attach to these final terms?

It will, I think, be of service towards the answer to these questions to begin by quoting some of the most pregnant passages from Hitzig and Munk. Owing to the wealth of the German language as regards psychological terminology, in comparison with English or French, the German interpretation has been by far the most searching, and by reason of this very wealth as well as by reason of the subtle harmonics and connotations in the individual words, that interpretation has remained practically ignored by us. Even Ferrier has not in his "Functions of the Brain," given at all an adequate account of Hitzig's view, still less of Munk's; Bastian ignores them both; and James, while giving abundant evidence of our great indebtedness to Munk, strangely enough goes the length of saying "that Munk's absolute tone about his observations and his theoretic arrogance have led to his ruin as an authority."

The most weighty critic of Munk is Goltz, less on account of his polemical skill than because of the weight of his experiments. We cannot but hesitate from an unreserved admission of Munk's data in so far as they have been derived from dogs, when Goltz brings before us dogs, first with one hemisphere removed, then with both hemispheres removed, apathetic and stupid it is true, yet able to feel, to move, to snarl and bark, to stand on their hind legs, to see and even to feed, and liable to have epileptic fits.[1]

[1] Goltz. Siebente Abhandlung über die Verrichtungen des Grosshirns. *Pflüger's Archiv.*, li. 1892, p. 570.

Wundt has recently published a critical review of Munk's doctrine of cortical projection in the 6th volume of his "Philosophische Studien," 1890. His criticism is particularly directed to Munk's retino-cortical scheme as being inclusive of two non-congruent conceptions of centralisation, relating (1) to distinctness of vision (sensation) (2) to complication of vision (perception). He characterises the view that high-level vision, hearing &c., are effected at distinct foci of visual, auditory, &c., areæ, as a revival of the phrenological faculty theory, and as leading into a psychologically untenable position.

But the question of interpretation may to a large extent be kept distinct from that of localisation; certain principles if true, are true whether cortical functions be localised or concentrated or diffused.

I have already on a previous occasion[1] pointed out the identity of idea between Munk's Seelen-blindheit and-taubheit, Rinden-blindheit and-taubheit, and Hughlings Jackson's well-known hierarchic scheme. And in translating Munk's language I shall take the liberty of rendering the German prefixes Seelen—and Rinden—by the English prefixes higher-level and lower-level, a rendering which appears to me to be less question-begging than a literal translation of these two very heterologous words, and justified by what Munk himself saysof th esense in which he uses the terms.[2]

Hitzig[3] says:

"A portion of the convexity of the dog's cerebrum is motor (using this expression in Schiff's sense), another portion is not motor."

"The motor portion, speaking generally, is situated anteriorly, the non-motor portion posteriorly."

"By electrical excitation of the motor portion one obtains combined muscular contractions of the opposite half of the body."

"With very weak currents the muscular contractions can be localised in definite and narrowly restricted groups of muscles. With stronger currents excitation of the same or of closely adjacent spots, causes other muscles, and even muscles of the same side of the body to participate in the contraction. The possibility of isolated excitation of a definite muscular group,

[1] "An Introduction to Human Physiology," 1891, p. 533.

[2] "Ich habe diese Bezeichnung im Jahre 1877 nach vieler Ueberlegung gewählt und ihr vor "Vorstellungsblindheit" oder "Erinnerungsblindheit," die am nächsten lagen, aus guten Gründen den Vorzug geben zu müssen geglaubt. Indem ich wiederholt definirte: Seelenblindheit=Fehlen der Gesichtsvorstellungen, der Erinnerungsbilder der Gesichtswahrnehmungen, dürfte ich die Benützung von "Seele" für gerade so unbedenklich halten, wie wenn ich α Blindheit oder β Blindheit gesagt hätte. Dass trotzdem Missverständnisse von seiten flüchtiger Leser nicht ausgeblieben sind, kann mich um so weniger veranlassen die Bezeichnung aufzugeben, als ich eine bessere noch bis heute nicht gefunden habe." *Munk*, Gesammelte Mittheilungen, 2nd edition, note to p. 41.

[3] Hitzig. Untersuchungen über das Gehirn. *Berlin*, 1874, p. 11.
Fritsch and Hitzig. *du Bois-Reymond's Archiv.*, 1870, p. 310.

using very weak currents, is, however, limited to very small spots, which for the sake of brevity, we may designate as centres."

"It is an assured fact that a considerable portion of the nerve-mass forming the cerebral hemisphere (say nearly one-half) is in immediate relation with muscular movement, and that another portion, at least directly, has no evident connection with it."[1]

"If we raise the question whether our excitation effects were caused by a direct action upon particular centres in which the voluntary motor impulse arises, or whether they are attributable to an excitation of subjacent fibres, or whether any third possibility exists, our answer must be far more reserved."

"Even if we assume as proved, that the movements in question are liberated by the ganglionic substance—and it is not proved—this would not be enough to prove that with these movements which are liberated by the internal event, that precise portion of the cortex furnishes the substratum of the first outward step in the series beginning with the origination of a sense-impression, and finding a temporary end in the expression of will evidenced as muscular movement."

"It is, on the other hand, far from inconceivable—and the notion is favoured by our knowledge of the anastomotic structure of these parts—that that portion of the brain which includes the birthplace of a will to move, is of another, or perhaps of a more complex nature, and that the parts which we have called centres only constitute agencies, exchanges, in which an arrangement of muscular movements occurs, similar to that effected through the grey matter of the spinal cord and basal ganglia, but more purposeful."

And later (page 30) describing the state of the two dogs upon which circumscribed portions of the brain (the "centres" for the anterior extremity) had been extirpated.

"Both animals, after extirpation of a portion of the (by us) so-called centre for the anterior extremity, had only partially lost the power of moving that limb, and had apparently suffered no loss of sensibility. But they evidently had only an imperfect knowledge of the state of this limb; they had lost the power of forming to themselves complete representations of its states; they were thus the subjects of a symptom, which appears

[1] Untersuchungen, p. 25-26.

in a very similar fashion in certain manifestations of tabes, only in this case, there was certainly no lesion of a sensory tract. One might express himself thus to characterise this condition more closely. Some kind of motor communication still persisted from mind to muscle, whereas, somewhere in the connection from muscle to mind there was a break. Possibly this break affected the terminus of the hypothetical path of the muscular sense, in any case it coincided with the position of the injured centre."

"Speculations concerning cerebral and mental activities have deservedly fallen into such discredit, that one may wisely observe an excess of caution in dealing with such matters."[1]

". . . We have, hitherto, avoided committing ourselves to a definite opinion whether we excited cells or fibres, termini or stations in the psychomotor circuit, and we shall still refrain from doing so. On the other hand, we have proved that the stimulus did act through known reflex channels. But if anyone should conclude, on account of these differences [polar differences, length of contraction] that the parts originally excited were not central expansions of the motor nerves, he clearly would have no grounds for the conclusion. For there is nothing to prove that central fibres (or cells) in connection with peripheral fibres (or cells) must react precisely like these peripheral elements."

"On the contrary, it is probable from all that we know that they react differently. And finally, the effects we have described have no analogy whatever among peripheral phenomena. One must, indeed, rest content with the assumption that the peculiarities depend upon the peculiar properties of the central organ."

". . . And yet I regret to have used the expression 'muscular sense,' for it has caused all kinds of misunderstanding from the very first. Nothnagel has, meanwhile, characterised the symptoms in question as disturbances of 'muscular sense,' far more definitely than I have. This determines me to specify my own conception more precisely, although I am most unwilling to depart from the reserve that I have imposed upon myself. For dealing with these factors as they become better and better known to us, an unknown magnitude and one which can hardly be ignored forces itself upon us—the Will. We stand towards this factor, as we do towards the elementary forces; we may know the modes of its manifestation, but not its essence, nor its actual nature."

[1] Untersuchungen, p. 52-53.

"We characterised the state of the animals we had operated upon as follows: They evidently had only an imperfect knowledge of the state of the affected limb; they had lost the power of forming complete representations of it; and we were justified in this conclusion by the analysis of the perturbations of movements that existed after destruction of that spot of the cortex, the excitation of which had caused movements of the now abnormally acting muscles. The question now before us may be thus simply formulated."

"Is the excluded centre the organ which alone governs the movements in question, or is there in addition to the general organ of will—assuming such to exist—another central motor organ in the same circuit of discharge?"

"To be as brief as possible we may refer to common experience of moderate movements by normal muscle. It is known that the image of movement caused in our own consciousness by muscular action is exceedingly clear. An expert painter would for instance be able to faithfully reproduce a posture assumed by himself with closed eyes. Nevertheless consciousness knows nothing of the moving factors; even the representations obtained by direct observation or otherwise, are so intimately connected with another department, that they contribute but little to the perception of motor forces regularly operating at the periphery. These are indeed scarcely known to us as regards each particular form of movement, they are only discoverable by inference; and digital palpation, *e.g.*, is far more helpful to this end than the repetition of the movement."

"Still it is clear that very exact representations of the state of muscle must be produced—as we learn from these very images of movement—and it is equally clear that these images are attributable chiefly to the perception of the muscular state, and only in minor degree to joints, skin, &c.: this we learn from the well known illusions of movements occurring in the paralysis of ocular muscles."

"If nevertheless our representations of the muscular state of our own body do not overstep the threshold of distinct consciousness, and thus enable us to see into the true nature of the process, we must attribute this failure to a very general law. We are able to distinguish the state of particular organs only in so far as is necessary and sufficient for their use in the uninterrupted maintenance of their function."

"But within such limits, the apprehension of these mainly unconscious representations of each particular phase of move-

ment, constitutes one of the necessary conditions of a normal progress of its succeeding phase; subsequently, (considering apparent muscular repose as a phase of movement) one must recognise that muscular states in general are among the various causes that guide the organism in its voluntary movements, and that regulate these movements. Let us assume an entire absence of all other sensory stimuli and perceptions, so that we have to do with a simple motor machine of such a kind set in motion by the muscular impulse, we may then very well imagine it as sufficient for the execution of purposive movements."

"We have recognised in the above described portions of cortex an organ the function of which coincides with that aspect of the psychical phenomenon that we have been considering, and I do not see the necessity for admitting that Will as such involves a specific and different motor organ. When in consequence of the joint action of several sensory impressions, either present or past, the motive of a movement arises, such motive never takes form thus: "Innervate muscle a b c, so that arm n, shall move through angle x," but thus: "take," "write," "speak," &c. The organs we are acquainted with, appear to me sufficient to enable us to form a conception of the normal progress of the movement when once it has been thus initiated. Points of detail are it is true still sufficiently obscure." (Untersuchungen, p. 59-61).

Munk[1] in his third communication to the Berlin Physiological Society says:—

"I have now to direct your attention to a third department of the cerebral cortex of the dog—the portion CDE, Fig. 1. You recognise at once that it is the portion of the cortex upon which so much consideration has been bestowed during the last eight years, since the first investigations of Fritsch and Hitzig. If, nevertheless, a clear understanding has not been arrived at as regards the functions of this cortical department, this must be attributed to the fact that we do not yet possess a guiding point of view for a correct appreciation of the experimental data. The further I advanced in the knowledge of the auditory, and more particularly of the visual region, the more orderly became data that appeared at first irreconcilable; and if last year I was obliged to content myself with presenting to you our cortical department as the motor area in contrast with the posterior sensory area, now I believe myself able to invite you to a deeper comprehension of this cortical region as well."

[1] Munk. *Du Bois-Reymond's Archiv*, 1878, p. 162.

"In a word our cortical region CDE is the sensory area (Fühlsphäre) of the dog. Just as the cortex of the occipital lobes stands in relation to vision, and that of the temporal lobes to hearing, so the cortex of the parietal area stands in relation to common sensation (Gefühlsinn); in this as in the other cases we have the locus where perception is consummated and where representations, the memory images of perceptions, have their seat. Let it be clearly understood, however, that it is not sensation of the skin only that is here in question, but sensation in its broadest sense, the sensation of the whole body." (p. 171.)

". . . After extirpation on a dog of a portion of cortex in the area CDE of one hemisphere, the sole and regular consequence (when inflammatory reaction has passed off) is disturbance of motility and sensibility on the opposite half of the body, in one part or in another according to the situation of the extirpated spot. With the lapse of time these alterations gradually vanish, sometimes less, sometimes more completely, so that finally in the most favourable cases our dog recovers the demeanour of a normal dog."

". . . Still these disturbances are very imperfectly characterised by the terms, disturbance of motility, disturbance of sensibility." (p. 173).

Then follows a careful description of the state of matters in a representative dog having suffered extirpation of D on left side, three to eight days after operation.

"The cortex of the parietal lobes of the dog is the sensory area of the opposite half of the body, and is composed of a number of regions each of which is related to a particular part of this half. In the percipient central elements of each region terminate contiguous fibres that subserve cutaneous, muscular and neural sensations (Innervationsgefühle) of each corresponding part of the body. Within each such region the sense-representations (Gefühlvorstellungen) of each such part of the body have their seat, so that the former constitute the special sensory areæ of the latter, *e.g.*, of the anterior or of the posterior extremities. Within the province of each such sensory area of particular parts of the body, small extirpations entail partial loss of sensory representation of the part, larger extirpations its complete loss—high-level paralysis (loss of high-level motility and of high-level sensibility) of the part; but in any remaining parts of a sensory area there can be a restoration of sense-representation.

By still more extensive extirpation, low-level sensations may be impaired, and only a portion of high-level sensation may be recoverable. The impairment and the failure of restitution are the greater, the smaller the remaining sensory area. Its complete destruction on one side has as its necessary consequence permanent loss of all sensation—low-level as well as high-level—total paralysis (total loss of movement and of sensation) of the related part of the body." (p. 176.)

". . . Perceptions and representations in the domain of common sensation (Gefühlsinn) have been so variously characterised and defined by various authorities, that in the absence of a particularly solid groundwork, our study of the sensory area would have been completely fruitless. I have, therefore, prefaced our consideration of the sensory area by a survey of sensory perception, by going over the organs of the body, the nerves of which subserve perception, and from which perceptions evolve representations. Still, in the consideration of neural sensations, I have restricted myself to defining them as perceptions derived from motor impulses accompanying active movements; it thus remains to be added what are the organs, of which the modifications can reach consciousness as neural sensations. These organs, as I have stated above, are the subcortical ganglia or centres in brain and cord, that invoke movements. As in the infancy of the animal, the representations of movement are developed from its first purely reflex movements; as in the adult animal, the representations of movements of a part of the body, can still arise in its sensory area, even if (*e.g.*, in locomotion), this sensory area is not actually participating in the accomplishment of such movements; as finally, the representations of movement lost in consequence of cortical extirpation in a given part of the sensory area, may be reformed out of the reflex and automatic movements of the affected part, it cannot but be that fibres ascend to the cerebral cortex from subcortical motor centres or ganglia, as well as from the skin and from the muscles—fibres that subserve cortical perception of these subcortical centres."

"Our neural sensations are indeed, for the present, to be distinguished from what has hitherto very generally received the name of neural sensation (Innervationsgefühl)—from the "perception of the intensity of voluntary effort in connection with voluntary movement. "Will," "voluntary movement," with seat and origin in the cerebral cortex, are indeed very convenient, and may, therefore, also be valid expressions, but they are destitute

of a phenomenal physiological substratum. All we know of the cerebral cortex is that it is the place of perceptions and the seat of representations. Beyond this, it is merely admissible to assume as has been assumed by Meynert on somewhat different data, and by Wernicke, that representations of movement are the causes of so-called voluntary movements; that with the production of such a representation to a given degree (and indeed with a production *viâ* association, not *viâ* its ordinary constituent sensations) *eo ipso* the corresponding movement is elicited; and that the greater its formative representation the greater the resultant movement. The "perception of the intensity of voluntary effort, in connection with voluntary movement," might indeed be the attribute of a representation of movement; a true perception might still obtain only indirectly, quite independently of the "Will," and the percept would then be nothing but the neural sensation in the sense just set forth." (p. 177-8.)

§2. Sensory and Motor Centres.

"Sensory" and "Motor" are used almost antithetically. "Sensory" connotes *physically* centripetal changes ending *subjectively* in feeling. "Motor" connotes *physically* centrifugal changes beginning, according to some thinkers spontaneously in a motor centre, according to others consequently upon previous contripetal changes.

I need hardly say that I adopt the second view, every centre must be sensori-motor, *terminus ad quem* as well as *terminus a quo*; and in the two different components of the double term I do not recognise any phenomenal division of the central process into sensory and motor. Strictly I picture one undivided change taking place in any centre, resulting from impulses to it and resulting in impulses from it; therefore as far as possible I avoid the term motor, believing that it is apt to imply the spontaneous origination[1] of force, but if speaking loosely in accordance with custom, I occasionally use this term and refer *e.g.*, to a motor area, I feel it impossible not to add a guarding phrase to exclude that crude and (to me) inconceivable implication. Between centripetal and centrifugal impulses, I see a single physical process, one

[1] This is explicitly declared in Bain's theory of Spontaneity.

and indivisible; to call it sensory or to call it motor, or even to call it sensori-motor, are to my thinking, imperfectly and improperly to designate it by more or less subjective terms—with more or less obstructive connotations. All that I can recognise in the notion of a centre is an organ of elaboration receiving and giving out impulses. By the term motor I denote that it emits but fails to denote that it receives; by the term sensory I denote that it receives (and imply that *It* feels) and fail to denote that it emits impulses; by the term senso-motor I denote reception, "feeling," and emission. All these meanings when closely analysed are illegitimate, and convey too little or too much. Experimentally I may not predicate "feeling" of any centre, but only of the hypothetical *ego*, I may only infer from visible movement that other animals "feel" and that sensations similar to my own are associated with the activity of certain centres.

This inference is unavoidably included in the word "sensory" every time we use it, and it is only in the loose language of the laboratory that we have any justification for speaking of a "sensory centre." We are always in presence of a process manifesting itself by movement or by want of movement, and although we may for practical purposes say in the laboratory that such a centre is sensory because after its removal signs of sensation have vanished, or motor because after its removal paresis has resulted, we have no strict logical right to say that a centre is sensory, or that it is motor, nor even that it is sensory and motor. We are in presence of a central process associated with centripetal and centrifugal processes, and we have no right to say that the centre is motor, or sensory, or both, otherwise than inferentially, and guarded by a clause of interpretation to indicate that we are conscious of the inference included in the term. The inferences contained in these qualifying adjectives are at once recognised by placing oneself in the critic's attitude. How do you know that the central process is motor and not sensory? Answer—By the movements which are its consequence, and by the absence of movements which I have learned to be expressive of sensation. How do you know

that the central process is sensory and not motor? By the movements which are its consequence or by the absence of movements which are the consequence of its annihilation, and here we clearly see that the process if sensory must also be motor, directly or indirectly, if it is to manifest objective signs. How do you know that it is sensori-motor? Motor by its movements, sensory of necessity. But in this answer we finally see that if motor may be predicated of a process seeing that it is the antecedent of motion, the word does not predicate enough, while to add "sensory" predicates too much.

So that in sum we may say—the central process is motor, *i.e.*, it produces movement; it must have centripetal antecedents, *i.e.*, motion is not spontaneously generated; it may be sensificatory, *i.e.*, a conclusion inferred from the data *sensatio mea et motus tuus*. "Sense sure you have, else could you not have motion," and we may find it convenient to use the term motivity or senso-motivity to designate this property of certain nerve-centres.

Is it not plain that inference from experiments as to motor and sensory centres and processes must have much deceptive verbal connotation to very little phenomenal denotation? What are the phenomena denoted by the "sensory centre" of a dog or the "motor centre" of a monkey, by a sensation, by an "afferent centre"? And how slight have often been the phenomenal data from which it has been said that this animal has lost such and such a sensation, that such and such a function is *localised* in this or that part of the brain. I understand the phenomenal meaning of an afferent tract or of an efferent tract, but not that of a motor centre or a sensory centre. I picture a wave of change passing through a cell, but do not know at what transverse section of the wave to label it motor or sensory. The property of cortical grey matter is sensomotivity; the most typical so-called motor cortex is senso-motor, the most typical so-called sensory cortex is senso-motor.

This has been no merely verbal disquisition, but on the contrary is fundamental to the subject. We must recognise that one indivisible *thing* is cut into two ideas by these *words*

"sensory" and "motor." A central process is not sensory or motor but senso-motor (in a guarded sense), and a centre is an "organ of return of action."

Let us apply this notion to the central process which we consider as associated with muscular movement, and see by its aid what we are to understand by such expressions as "motor idea," "idea of movement," "kinæsthetic idea."

By those who make a distinction and contrast between a motor and a sensory process, the motor idea is a mental picture of *movements about to be made*, the kinæsthetic idea is a mental picture of the results of *movements just made*. The motor idea is considered as an antecedent to motion, the sensory idea as its consequent. It does not matter whether we further admit that we may have the idea of movement without actual movement, or as the antecedent of a future but deferred movement, or as the consequent of a movement made long ago, we must in all cases recognise that it is illegitimate to say that the idea of movement is exclusively a motor idea, an image of an imminent motor discharge, or that it is exclusively a sensory, *i.e.*, kinæsthetic idea, an image of the results of previous similar movements. It is both. Each movement is an element in a series of movements, its idea or image is at once the consequence of past movements and the antecedent of the movement in consummation, which movement in consummation as fast as it progresses is attended with renewed impressions from the periphery pouring into the sum of past sensation at the centre. In a movement made at this moment, the image of the past similar movements is at the same time the image of the actual movement, which by its results repeats a similar image guiding the movement itself in its progress, and educating the centre towards future similar movements. It is not possible to distinguish a "kinæsthetic" image of past movements from a "motor" image of impending movements. The two words denote one thing.[1]

[1] Thought of movement is memory of movement, or more generally the *thought* is the *remembered*. An impulse, an intention, a resolution, a prayer, is a more and more concentrated act of attention, of memory, of thought. An identical neural process is the essential phenomenon wrapped up and presented in these very different words, will, attention, memory, belief, thought.

Nevertheless we can and do recognise in the use of the word senso-motor, which connotes the notion that the centripetal generates the centrifugal phenomenon, the principle that phenomena have generators and consequents antecedents; we are reminded by the word that a centre is an organ of return of action and that the type of all motor action is a reflex act. This principle is recognised by all leading workers and thinkers—by Hitzig in his conception that the motor area of the cortex is a "muscular sense" area; by Munk in his conception that it is the motor "Fühlsphäre;" by Bastian in his conception that it is the centre of "kinæsthetic" impressions. These three conceptions are but one and the same conception, which I most explicitly and unreservedly accept as a fundamental article of thought.

But beyond this fundamental and simple article that all action is literally "ex-cited," that any centre is an organ of return of action, or more generally still, that as far as our experience goes all phenomena are generated, not self-created, the ways part, and in the further description of the neural government of the body, which various observers and thinkers feel themselves entitled to give, variety and contradiction arise. Yet neither a purely motor view on the one hand, nor a purely sensory view on the other, can constitute for us a complete and satisfactory theory; each view is true in so far as it contains half the truth, but neither is true in so far as each contains only half the truth; the whole truth is contained in the sum of both views. The Rolandic area is senso-motor. And in the mental picture that we form of the neural government of the body, it is not sufficient to admit by itself, either the undoubtedly true principle that all motion immediately or remotely is an outcome of centripetal constitution, or the equally undoubted truth that the cortex is "motor" in so far as its excitation immediately or ultimately produces motion—but we must also admit (as I have urged elsewhere) that "in the co-ordinate function of any synergic group of muscles at least three conditions must be fulfilled: (1) impulses must be emitted from the centre in appropriate manner and degree; (2) the muscles must be 'in touch' with the spinal

axis, duly responsive to the reflex ebb and flow arising from altering peripheral states; (3) the muscles must be in touch with each other, directly reacting against that passive extension which antagonist muscles undergo from each other."[1]

It will however be convenient to postpone further consideration of this view to a later stage.

§ 3.—Cortical Centres.

A little reflection and a very slight acquaintance with "la belle doctrine" of cortical localisation, as that doctrine has been stated and supported and controverted by various authorities and writers, will very adequately correct any disposition we may feel to regard the examination of the terms used as a barren dialectical exercise. The differences of view have involved matters of fact far less than inferences from fact as embodied in words; identical words having been used of very unequal phenomenal value, and similar phenomena having been dressed up in words of apparently widely differing and even contradictory meaning. The effects of excitation and of lesion in the Rolandic area evidently have not been and cannot have been of such different phenomenal value under the hands of Fritsch and Hitzig, Ferrier, Munk and Goltz, as seems to be indicated by the terms "muscular sense centre," "Fühlsphäre," "motor centre," "sensory centre."[2] It can only have been by a defect in the valuation of words that we have had presented to us denial of localisation, in presence of descriptions of "motor" and "sensory" centres as definite as descriptions of carrots and of turnips.

The form which the verbal controversy has taken in this country is represented with greatest clearness and

[1] Waller. "Physiology," p. 466, also pp. 341-2.

[2] I need hardly point out again that my paper does not directly involve any question of actual localisation. Its main arguments are equally applicable, whether "sensomotility" be the property of the entire cortex or of only its Rolandic area, whether we admit motor and sensory centres as taught by Hitzig and by Ferrier, or the cortical map of Munk, or indifference of function in the unrestricted sense of Flourens, or in the restricted sense of Goltz.

intensity by the Ferrier *v.* Bastian issue concerning the functional significance of the Rolandic area.

In the early days of cortical experimentation Ferrier designated the results of cortical stimuli as "motor." He says:[1]

"By the term motor centre, as applied to a particular "part of the cortex of the brain, I have endeavoured to signify "the fact that this part is in direct communication with the "motor tracts and their ganglia, and that its function is to "excite co-ordinated muscular action of a definite kind of the "nature which we call voluntary."

Here and elsewhere Ferrier defines the sense in which he uses the term "motor," and having done this is legitimately entitled to use the term to designate as "motor," those parts of the nervous system which he finds to be concerned in the production of movements.

But Bastian, confining his attention to Ferrier's experiments, altogether objects to the word "motor," emphatically denies that the Rolandic area is motor, and designates it as a "Kinæsthetic" area. It is not difficult to see how this apparently fundamental difference has arisen. Ferrier had in view movement and its central origin, and used the term motor as if implying the spontaneous origination of motor impulses in supreme psychical centres; he nowhere defends himself from the truism that phenomena cannot, as far as we know, be spontaneously generated, and that what we call a "motor" impulse is necessarily not an uncaused motor impulse, but a resultant of antecedent phenomena; indeed he appeared to adhere to a view of central initiation by the introduction of the somewhat unfortunate term "psychomotor." He may or he may not, consciously or unconsciously, have been influenced by the well-known Aberdeen doctrine of motor spontaneity as taught by Bain. In so far as Bastian entered a *caveat* against this neat "motor" doctrine at a time when it was dominant in this country, he rendered good service to our true knowledge of the subject. And that service would have been still more complete if it could have been made plain to us that kinæs-

[1] West Riding Lunatic Asylum Reports, 1874, p. 47.

thesis implied neither more nor less than is expressed in the analyses of Meynert, Wernicke, Schiff, Fritsch, Hitzig, and Munk. Then the turn might have been understood and welcomed as the expression of a familiar German postulate, viz., one indivisible process—kinæsthesis, ideally composed of æsthesis leading to kinæsis. If that is the sense in which Bastian has used the term, I unreservedly accept it as denoting a fundamental postulate.

The phenomena antecedent to "motor impulses," (leaving on one side effects of nutrition or injury or experimental stimuli), are what are commonly designated as sensory, or more properly centripetal phenomena. Whereas Ferrier with his attention focussed upon the effect, calls the centre to which he traces it "motor," Bastian goes back a step further and refers the muscular movement to its centripetal antecedents which he designates "kinæsthetic." These antecedents are for him so striking that he denies their central resultant to be motor; yet he recognises that centrifugal processes occur along efferent tracts (which he designates "internuntial") connected with the Rolandic area, and it is plain that in denying that that area is "motor" in Ferrier's sense, all that he really denies is that it is *spontaneously motor*. He contradicts "la doctrine brutale" of motor centres.

If anyone can doubt that this is the correct interpretation of Dr. Bastian's position concerning the Rolandic area, his mind will be set at rest by referring to the diagram, in which Dr. Bastian has represented the physiological anatomy of the "kinæsthetic" process. (BRAIN, 1892, p. 31. Fig. 1.)

He has there made the matter perfectly clear, and in so doing has shewn how unnecessary it has been to burden and obscure his presentation of so simple a matter by such terms as "kinæsthesis" and "internuntial." The facts presented are admitted by everybody, the words in which they have been clothed are mystificatory. And the principal part in this disguise is evidently that played by the word "internuntial" as applied to the pyramidal tract. Dr. Bastian's contention is that the pyramidal tract is not efferent but internuntial, that therefore the cortical area from which

it proceeds is not motor. In Dr. Bastian's diagram a Rolandic centre is pictured in connection with an afferent tract and an efferent (internuntial) tract, a spinal centre on the afferent tract is designated afferent, that on the efferent tract is designated efferent; but the Rolandic centre (by calling the pyramidal tract internuntial and not efferent) is made to be on an afferent tract only. Therefore the Rolandic centre is not "motor," the only "motor" centres are bulbo-spinal.[1]

That Dr. Bastian's views are principally if not exclusively determined in opposition to the views of Dr. Ferrier, is equally clearly indicated in this his most recent publication.

"The so-called motor centres of the cortex were not, of "course, originally supposed to be termini for afferent impres-

[1] It is surely inconvenient to speak of afferent and efferent centres, afferent and efferent impulses, and more verbally correct to speak of afferent and efferent tracts, centripetal and centrifugal processes. As regards centres the difficulty of qualification is greater. We may indeed speak of afferent and efferent centres when they are known to be on afferent and efferent tracts; but as regards cortical centres this cannot be done. "Motor," "Sensory," and "Sensori-motor" are the words which I find it necessary to use in elementary teaching, but they are not unobjectionable. The central terminus is *ad quem* and *a quo;* but whether the arrival and departure platforms are conjoined in one "Sensori-motor" centre or in "sensory" and "motor" centres commissurally joined, is a different question. The present tendency is to accept the "sensori-motor" character of the cortex in the sense indicated by Munk's account of his own experiments.

The truism that by sensory centre or motor centre we cannot mean a centre which feels or which causes motion, but at most a place where sensation occurs or a motor impulse is elaborated, is worth making particularly clear if only as a matter of definition.

I have two laboratories, in one of which I learn, while in the other I teach. The first is my sensory laboratory, the second is my motor laboratory, and the Edgware Road is the main internuntial tract between them. But the laboratories are themselves neither sensory nor motor, they are places in which sensation and motion are produced. And in this restricted sense they are not purely sensory, nor purely motor; the first is motor through the second, the second is sensory from the first.

One of my friends has a single large laboratory in which he learns and teaches. It is his senso-motor laboratory, and as a matter of convenience it is divided into rooms in which most sensation occurs, and rooms in which most motion occurs. But here again the rooms are not purely sensory nor purely motor.

Now *a priori* it can be that in that psycho-physical laboratory called the cortex cerebri there are sensory departments and motor departments. It cannot be that the cortical departments are purely sensory or purely motor, but to what degree, if any, sensation and motion and particular forms of either are concentrated or localised in particular departments can only be ascertained by the *a posteriori* method, by actual experiment.

But experiments are conflicting, Goltz's dogs absolutely contradict the dogs of Munk, and complete (*i.e.* logically insulated) data on the human brain are few.

"sions; when first discovered, they were said and they are still "maintained by Ferrier to be true "motor centres." (BRAIN, "1892, p. 30).

As a matter of fact, the excitable area of the dog's brain when first discovered by Fritsch and Hitzig, was by them inferred to be a "muscular sense area." Ferrier, while maintaining that the Rolandic area is "motor" inasmuch as it gives rise to motion, most assuredly would not assert them to be "true motor centres" in the absolute and exclusive sense implied by Bastian.

Goltz in his first[1] (and subsequent) papers on the brain, lays far more stress upon loss of sensation than upon loss of motion in consequence of cortical ablation.

Schiff[2] interpreted Hitzig's extirpation and excitation effects to signify that the cortex is organ of return, and lays stress on the loss of sensation due to extirpation.

The necessary functional connection between "motor" cortex and centripetal impressions (using "motor" in its carefully guarded sense to qualify a point of cortical departure and not a point of origination), is experimentally demonstrated in the observations of Bubnoff and Heidenhain.[3] Cortical excitations (dog) producing contractions after an interval of .03" to .04", did so during excitation of the central end of the divided sciatic[4] after an interval of .6" to .12". Here was a retardation of "motor" elaboration, due to centripetal impressions; under different conditions a precisely opposite effect of the same cause was observed, viz., a shortening of the reaction-time, *i.e.*, a sign of increased "motor" or centrifugal tension.

And the whole spirit and tendency of Munk's investigations have been in the broader sense which is thus entirely ignored by Dr. Bastian. From first to last (1877 to 1889) Munk, in his interpretation of his own results, holds prominently before us the "arrival aspect" rather than

[1] Goltz. *Pflüger's Archiv.* 1876, xiii., p. 9.
[2] Schiff. *Rivista Sperimentale*, 1876.
[3] *Pflüger's Archiv.* xxvi., p. 164, 1881.
[4] It is, perhaps, not superfluous to say that this is not quoted in support of any centripetal influence along an efferent tract.—*Vide infra*, p. 382 *et seq.*, and Bastian (BRAIN, 1892, p. 26, footnote.)

the "departure aspect" of cortical processes, so prominently indeed that he has given to the Rolandic department of the cortex the name of Fühlsphäre.

Professor James interpreting the physiological phenomena from a psychological and critical standpoint, comes to a very similar conclusion; he very happily compares the motor area to the "mouth of the funnel," through which pour outgoing impulses, caused by incoming impulses. His analysis also leads him to recognise the illusory character of a sharp distinction between motor and sensory centres.

". . . so that really it (*i.e.*, a 'motor' centre) is only the "mouth of the funnel, as it were, through which the stream "of innervation, starting from elsewhere, pours; consciousness "accompanying the stream, and being mainly of things seen "if the stream is strongest occipitally, of things heard if it is "strongest temporally, of things felt, &c., if the stream occupies "most intensely the motor zone."—(*Psychology*, vol. i., p. 65.)

I very cordially agree with this kind of verbal representation, distinctly adding, however, that I do not agree with the arbitrary and dogmatic exclusion of the motor zone from the "sphere of consciousness," elsewhere enunciated by Prof. James, and approvingly quoted by Bastian, Ferrier, Gotch, and Horsley.

"If the motor cells are distinct structures, they are as "insentient as the motor nerve-trunks are after the posterior "roots are cut. If they are not distinct structures, but are "only the last sensory cells, those at 'the mouth of the funnel,' "then their consciousness is that of kinæsthetic ideas and sensa-"tions merely, *and this consciousness accompanies the rise of "activity in them rather than its discharge.*"—(*Psych.* ii., p. 517.)

It is to the last sentence mainly, indicated by italics (not in original[1]) that I take exception. *Non sequitur.* The assertion is no more than an assertion; it is no necessary associate of James' second alternative, and, in my view, he has no right to thus distinguish between the "rise" and the "discharge," nor to attribute consciousness exclusively to either.

[1] In all other parts of this paper italics in quoted passages are italics in the originals.

"All paths are paths of discharge, and the discharge always "takes place in the direction of least resistance, whether the cell "which discharges be 'motor' or 'sensory.' The *connate* paths "of least resistance are the paths of instinctive reaction; and "I submit as my first hypothesis that *these paths all run one way*, "*that is, from 'sensory' cells into 'motor' cells, and from motor* "*cells into muscles, without ever taking the reverse direction.*

"A motor cell, for example, never awakens a sensory cell "directly, but only through the incoming current caused by the "bodily movements to which its discharge gives rise. And a "sensory cell *always* discharges or normally tends to discharge "towards the motor region. Let this direction be the 'forward' "direction. I call the law an hypothesis, but really it is an in-"dubitable truth. No impression or idea of eye, ear, or skin "comes to us without occasioning a movement, even though the "movement be no more than the accommodation of the sense-"organ; and all our trains of sensation and sensational imagery "have their terms alternated and interpenetrated with motor "processes, of most of which we are practically unconscious. "Another way of stating the rule is to say that, primarily or "connately, all currents through the brain run towards the Rolan-"dic region, and that there they run out, and never return upon "themselves. From this point of view, the distinction of sensory "and motor cells has no fundamental significance. All cells are "motor; we simply call those of the Rolandic region, those "nearest the mouth of the funnel, the motor cells par excellence." —(*James' Psychology*, vol. ii., p. 581).

I am again in agreement with this mode of view, and most unreservedly so with the concluding paragraph. But I would appeal to Prof. James himself, to bear witness to the inconvenience of thought and expression into which he must have repeatedly found himself forced by the use of these two words, "motor" and "sensory," with their implications of distinction between motor centres and sensory centres. I have elsewhere given expression to the same idea in the following terms:

"Any motor or discharging centre must also be a 'sensory' or receiving centre; it must be excited as well as excite. Any 'sensory' centre must also be motor, directly or indirectly; else we could have no objective tokens of sensation; every centre, whether called motor or sensory, is *terminus ad quem* as well as *terminus a quo*."

And with regard to the law of forward direction I fully agree with James that it is not an hypothesis but an indubitable truth, at any rate as regards efferent channels. That the vortical movement, of which organisms are the scene, and their nervous systems the highest organic expression, *should* exist in one direction is *a priori* probable. That the vortical movement *does* normally exist in one direction in the nervous system, is known to everyone who knows that stimulation of the central end of an afferent nerve, or of the peripheral end of an efferent nerve produces movement, while stimulation of the peripheral end of an afferent nerve, or of the central end of an efferent nerve produces no movement. Yet that it *may* possibly be produced in an opposite direction, at least in centripetal channels, is indicated by more or less convincing considerations, viz., by Du Bois-Reymond's negative variation in the posterior roots, and by Gotch and Horsley's observations of spinal discharge, *viâ* the posterior roots. Wundt indeed accepts the possibility to such an extent that he speaks of "sensory-centrifugal" channels and accepts without comment the view that a hallucinatory disturbance of the brain may propagate itself down the optic nerve to the retina.[1] But to weigh this doubtful point is foreign to my present purpose.

That an effective impulse cannot occur *up* an efferent channel, is proved by the absence of reflex action when an efferent nerve is excited.[2]

§ 4—Elementary Considerations.

I had fallen into the habit of imagining neural processes on a three-level scheme, before even knowing the name of Hughlings Jackson, and had adopted as typical diagrams of reference the two schemata (Figs, 1 and 2) which I now reproduce from notes put on paper in 1876, adding to them the expressions then used, and the Jacksonian expressions that I have since learned. But I have never regarded such a three-level scheme as anything more than a convenient

[1] *Physiol. Psychol.*, vol. ii., p. 432 (3rd. ed.) Engelmann's retino-motor fibres are not here in question.

[2] See also page 386.

scaffolding upon which to hang certain functional conceptions.

I shall give diagrams in the endeavour to employ every possible means of exhibiting the tenour and tendency of my mode of view, and in the belief that a presentation in black and white of the schemata and of the symbols which have served as a standard of meditation, will help to expose the drift of reasoning—necessarily in large measure psychological, and therefore liable to extravagance of abstraction.

A principle that has greatly contributed to my thought-guidance is the notion that every neural process, even the lowest, is on the type of the psychological process of inference. I have gradually associated this notion with doctrines proceeding from empirical psychologists, more especially from Wundt, Bain and Fechner;[1] viz., from Fechner the relation between stimulus and sensation, from Bain the law of relativity, from Wundt the doctrine of neural inference. Without undertaking an adequate (and therefore lengthy) exposition of the relation knitting together these several topics, I must now content myself with a bare indication of the kind of relation which appears to me to exist, enough to justify the schema of inference which has served as one of my guiding notions. I will do so as briefly as possible in the following paragraphs:—

It appears to me to be very essential to recognise the applicability of the law of relativity in the most elementary sensation as well as in the higher provinces of judgment. In Fechner's theory a sensation is a comparison between two stimuli; the proper symbol indicative of its objective composition, is therefore a ratio $\frac{a}{b}$, and not any absolute quantity a or b.

I have gradually come to adopt the logical expressions: *major premise*, *minor premise* and *conclusion*, in analysing the factors of the simplest sensation, as well as of the most com-

[1] I name only those sources from which I have consciously derived instruction. I notice that James states that Wundt and Helmholtz have more recently "recanted" (*i.e.*, retreated from the neural inference position). As far as my reading of Wundt goes, this seems to me a very gratuitous and misleading supposition on the part of Professor James. ("Psychology," ii. note to p. 111.)

plicated mental judgment. This is not intended to imply that the syllogistic terms of logicians correspond to any physiological factors in a neural process, but is rather the consequence of early associations, and of subsequent experience of the great convenience of this mode of formulation as regards the detection and localisation of fallacy in particular cases. And I think that the use of these terms is perfectly compatible with an arrangement of factors in the form of an equation, which although in substance identical with the three syllogistic propositions, appears to me more closely physiological than these as regards the representation of the neural process of inference.

But at this juncture it is of particular importance to exercise all possible care to avoid confusion between sensation—the subjective phenomenon, and sensificatory change of state, its imaginary objective substratum. And it is to guard against this fallacy that in symbolising the phenomena I find it useful to reserve Greek letters for the subjective and Roman letters for the objective state.

The simplest present sensation σ is not the concomitant of any isolate present state of organ S, but the resultant of a comparison based upon state now and state before now, *i.e.*, of a ratio $\frac{s}{s'}$.

The organic state now is the sum of many previous alterations of state; many sensations σ the concomitants of many previous ratios $\frac{s}{s'}$, may be conceived as summed up in the state of sensibility Σ.

This state of sensibility Σ has as its organic basis a material state, which (having regard to the total Σ by many elementary σ, each concomitant with a ratio $\frac{s}{s'}$) is properly represented by the ideal symbol $\frac{s}{s'}$.

Thus Σ the specific sensibility, the subjective resultant of past experience, is the concomitant of $\frac{s}{s'}$, the imaginary organic sum of the antecedent series of organic ratios $\frac{s}{s'}$.

To this resultant state (subjectively Σ objectively $\frac{s}{s'}$) I apply the term "personal ratio," meaning to connote in this expression that the personal state now is composed of compared objective phenomena and not of *absolute* objective

phenomena. Delbœuf has long since expressed this idea with the utmost clearness.

This personal ratio I take to be the subjective attribute of the organic *major premise*, each new sensificatory change of state I regard as a *minor premise*, and I consider that a *conclusion* is formed by the compounding of these two premises; this compounding or neural inference may, I think, properly be represented symbolically in the form of a multiplication, in which the product represents the conclusion, and at the same time the new major premise in relation to subsequent minor premises.

$$\frac{s}{s'} \times \frac{s}{s'} = \frac{s}{s'}\frac{s}{s'} \text{ or subjectively } \sigma.$$

But here it is necessary to justify the transition from the use of the word "ratio" to that of the word "premise," and the use of a fractional symbol for both words.

The terms of any premise or proposition are the subject and the predicate (joined by the copula); in a major premise the subject enumerates and the predicate denominates. The major premise "all men are mortal," may be written $\frac{\text{all men}}{\text{all mortals}}$ in which we enumerate "all men" as forming part of "all beings denominated mortals."

The minor premise "*Socrates is a man*" may be written similarly $\frac{\text{Socrates}}{\text{men}}$ by which we enumerate Socrates as belonging to the denomination men just enumerated in the subject of the major premise. And the syllogism may be written :—

$$\underset{\textit{major.}}{\frac{\text{men}}{\text{mortals}}} \times \underset{\textit{minor.}}{\frac{\text{Socrates}}{\text{men}}} = \underset{\textit{conclusion.}}{\frac{\text{Socrates}}{\text{mortals}}}$$

This is not an essay on logic, else we might proceed to urge that in a syllogism admitted to be valid by unanimous verdict, the major premise may be a truism, and the minor premise a clearly demonstrated fact; but I am at present only concerned to justify the indiscriminate use and identical symbolisation of a ratio and of a premise. The short account just given may suffice to indicate what I conceive to be the essential identity pervading the whole range of neural inference phenomena from the simplest sensation to the highest judgment.

But this description has only represented the process starting from given major and minor premises; it does not represent the mode of formation of a major premise, nor exhibit what in the process of sensation or judgment must be conceived as the order of procedure. A very few words will however suffice.

The starting point of the growth of sensibility is the ultimate fact "sensation," or the minor premise; sensibility or the personal ratio is the resultant of many past sensations or minor premises, *i.e.*, centripetal induction or synthetic generalisation of particular experiences. The starting stimulus in each act of sensation or of judgment is a sensificatory minor premise, and the consummation of a sensation or of a judgment is a compounding of premises in consciousness, attended with what can only be indicated as an intuition of parity or disparity between the psychological factors on the two sides of an equation, *i.e.*, major, minor on one side, conclusion upon the other.

I do not wish to press this symbolic mode of representation too far, yet I feel entitled to urge the picture as a useful one in bringing home to us the essential similarity of neural process concomitant with the whole range of subjective phenomena from the simplest sensation to the most complex judgment. It is in my opinion most congruent with the derivation of voluntary from reflex action, and with a physiological conception of the mechanism of ratiocination. It would be absurd to say that this or any other mode of symbolisation "explains" judgment, attention, sensation, but it is not too much to say that it clearly exhibits the possible factors of neural inference; each act of observation, each determination of conduct, is the resultant of two factors (1) the major premise or central state, more or less ad-tended to: (2) a particular minor premise or group of sensificatory stimuli, also more or less ad-tended to (within the remaining sensificatory field, less or more ad-tended to.) And in any theory of ratiocination, we must recognise that the primitive mental process is one of centripetal induction, its immediate consequent one of centrifugal deduction, and all higher mental processes on this reflex type compounded

of centripetal induction of minor premises with centrifugal deduction from a major premise.[1]

Considering the centripetal aspect of neural processes we may with advantage appropriate to the three-level scheme the three terms *impression, sensation, perception*—using "impression" as the lowest-level term, "sensation" as the middle-level term, "perception" as the highest-level term; and taking impression to denote an effect that does not reach consciousness, sensation (=empfindung) to denote a felt impression, perception (=wahrnehmung) to denote a sensation in its felt circumstances, *i.e.*, in relation with the group of associated sensations forming the mental picture of the moment (=vorstellung). The relative significance of these more or less abstract and figurative terms may be assisted by comparison with a more concrete case. Taking cerebral cortex as the analogue of retina: the mental picture or representation or cerebral field of attention is analogous with the retinal field of vision; the perception is the analogue of the distinct object in the centre of the field, sensations the analogues of surrounding objects within the field viewed indistinctly; impressions the analogues of surrounding objects in the field that are not seen, although they can be seen. This indeed is rather more than an analogy, there is actually an association between physiological field of vision and psychological field of attention; retinal field is really retino-cerebral field; the great psychological importance of visual investigation is attributable to this association as well as to the fact that the visual field is the more complex and more susceptible of experimental analysis than any other of our sense fields.

[1] Readers of Spencer will probably recognise the close similarity of this mode of representation with that contained in the "Principles of Psychology," vol. ii. I have not consciously borrowed them from that source, yet had I not there found the equation between two ratios urged as a more natural mode of representing ratiocination than the syllogistic form of logicians, I do not think I should have ventured to promulgate such a symbolism, although strongly persuaded of its greater fidelity to physiological phenomena. "Every ratiocinative act is the indirect establishment of a definite relation between two things, by the process of establishing a definite relation between two definite relations."—Spencer's "Psychology," vol. ii., p. 16.

I have long been in the habit of making use of the following table and schemata, and have found them of service in guiding my thoughts. I have refrained from publishing them, believing the considerations embodied in them to be admitted as truisms, and feeling no call to add another verbal skeleton to the literature of the subject. But having used them and finding that they have led me to certain peculiarities in the mode of view, I find it necessary to expose them to criticism, as forming an introductory basis of what I believe to be a (in some degree) rational reading of cerebral psycho-physiology.

GENERAL ATTRIBUTES OF THE CEREBRO-SPINAL CENTRE.[1]

I.—REFLECTION.—Every central element is an organ of return of action. It is excitable-exciting.

II.—RESISTANCE.—Each central element has a specific capacity for centrifugal tension resultant from centripetal impressions.

III.—RADIATION.—Centripetal impulses to a central element radiate to connected elements.

Inferior impressions influence superior centres; superior expressions influence inferior centres, in accordance with degree of resistance and strength of impulse.

IV.—RETENTION.—After each period of activity and restoration there remains an altered constitution of central elements, in which resistance to homogeneous activity is diminished, to heterogeneous activity increased.

These four R's embrace all the phenomena that came under my consideration, but I subsequently supplemented them by the following article, which is in reality an amplified statement of Article IV. The fundamental phenomena of exhaustion and replenishment (although constantly effective conditions of nervous processes) did not appear to me to belong to the present order of considerations, and are therefore not formally acknowledged here.

V.—SUMMATION (AND INHIBITION). — Two or more impulses summating in centrifugal tension or expression

[1] Each of the propositions enunciated on this page is considered to be preceded by the words "It is conceivable that . . ."

may be individually "friendly" or "unfriendly" to one another.

Homogeneous impulses or groups of impulses received simultaneously or successively by the same central elements produce a sum of effect greater than the effect of a single impulse or group of impulses—*i.e.*, are "friendly."

Heterogeneous impulses to the same central elements produce a sum of effect smaller than, or different from, the effect of a single impulse—*i.e.*, are "unfriendly."

Any two or more impulses or groups of impulses, whether homogeneous or heterogeneous, successive or simultaneous, upon different central elements, may be friendly or unfriendly; they are more commonly unfriendly.

The terms "impression" and "expression" are used above in a very general sense to denote centripetal impulses from the periphery towards a centre, and centrifugal impulses from a centre towards the periphery.

The impression to a superior centre is constituted by the impressioned resistance of an inferior centre.

Given three centres ranking I., II., III., and indicating impression, expression, and alterations of resistance or tension by appropriate letters and numerals. An impression I_3 enters III., produces an alteration R_3 and liberates E_3. Altered R_3 constitutes impression I_2, I_2 enters II., produces an alteration R_2 and liberates E_2. Altered R_2 constitutes impression I_1. I_1 enters I. alters R_1 and liberates E_1. A second identical impression I_3 will meet with less resistance in III., it will produce a smaller change R_3 and conceivably a larger liberation E_3; a smaller change R_3 will constitute a smaller impression I_2, therefore a smaller change R_2, therefore a smaller impression I_1, &c.

As the consequence of repeated identical impressions we thus have :—diminished resistance, the diminution being greatest in III., less in II., least in I.; diminished impression, the diminution of I_2 being greater than that of I_1; increased expression, the increase being greatest of E_3, less of E_2, least of E_1, or otherwise: the minimum effective impression diminishes with repetition; the diminution is greatest in the case of I_3, less in that of I_2, least in that of I_1, and we have as the

final result of repeated activity, greater diminution of R_3 than of R_1, and a scale of most diminished minimum I_3 to least diminished minimum I_1 (with constant expression) of most increased E_3 to least increased E_1 (with constant I_3); which I take as signifying how, *With repeated exercise the reflex function specific to each centre, ranges in degree from most perfected in* III. *to least perfected in* I.

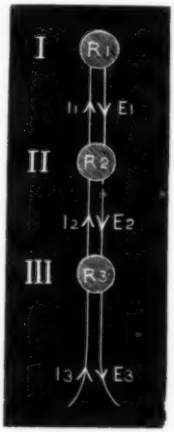


FIG. 1.

It involves these three assumptions:—resistance diminishes with repetition; impression ascending from lower to higher centre varies in the same sense as the resistance overcome in the lower centre; expression descending from higher to lower centre, or from lowest centre to periphery, varies in an opposite sense to the resistance overcome in the expressing centre.

The terminology used may prove a stumbling block to the word-picture I am attempting to compose. Impression and Expression are used in a peculiar and most general sense to denote impulse *to* and impulse *from* a centre. The diagram will, doubtless, make that sense plain. It contains this ambiguity. In the case of centre III. for instance, we have, properly speaking, two lines of expression *from* that centre,

one E_3 towards the periphery, the other I_2 towards the centre above. And to the same centre III. we have *two* lines of impression, one I_3 from the periphery, the other E_2 from the centre above. Thus I_2 is expression with reference to III., impression with reference to II.; E_2 is expression with reference to II., impression with reference to III. A similar ambiguity attaches to I_1 and E_1. But it was necessary to make choice of symbols, and this has been done in accordance with direction to or from the highest centre.

I have employed the physical figurative term *resistance* after some hesitation, but no distinctly preferable term offered itself. Perhaps the words tension or elasticity come closer to the idea intended to be conveyed. We may imagine a centre as an elastic reservoir of tension, in which an ingoing impulse may be absorbed with increase of tension which may or may not express itself in an outgoing impulse. But to this physical term we must attach chemical connotations, considering that the impulse elaborates greater chemical instability or actual discharge. Thus "resistance" is equivalent to chemical "stability," diminution of resistance is diminution of chemical stability. But what now do we mean when we say that an impulse in passing through a cell "meets with less resistance," or produces a smaller alteration of tension, and how can we picture this in chemical terms? Such modes of expression obviously give a most imperfect picture of what actually takes place. An impulse does not properly speaking *pass through* a cell, it evokes elaboration within the cell, and in this view resistance is chemical inertia; but diminished resistance is not diminished chemical inertia, it is diminished chemical change or diminished chemical friction. I have said enough to indicate the imperfection of idea which is apt to be connoted by either a physical or a chemical figure of speech. Yet I have perforce made use of such a figure, viz., "resistance," choosing it as the most familiar, but guarding myself from the appearance of pushing the physical analogy suggested in the word.

The effects of an impulse upon a cell (indicated by the word alteration of resistance, and in the diagram by the letters R_1 R_2 R_3) I picture to myself somewhat as follows—

taking for illustration the centre III. The impulse I_3 influences cellular energy; it increases[1] potential energy, it may render potential energy actual; it tends to overcome or actually overcomes inertia, and in the latter case it effects friction. The actual effects of this cellular elaboration are the impulses E_3 and I_2. In saying that a second impression I_3 meets with less resistance, I imagine that the cell matter is more unstable; this would seem to imply that the impulse would more easily disturb the whole mass of the cell, producing a more extensive change and thereby greater E_3 and I_2. But I do not admit this. I imagine the greater instability of the cell to be *molecular*, so that the disturbance by a second impulse can propagate itself with less diffusion, overcoming less chemical inertia, and producing less friction, than the disturbance produced by a first impulse. The analogy with electrical resistance may make this point clear, but it is only an analogy. An electrical current entering by one point of a wet sponge of lower resistance would propagate itself with less diffusion, overcoming less chemical inertia, and producing less friction than in the case of a wet sponge of higher resistance.

Thus it is that I am led to imagine that the disturbance of a nerve-cell by a second impulse I_3 gives rise to a *greater* impulse E_3 and to a *smaller* impulse I_2, than did a first impulse I_3.

First impressions are the most intense in consciousness. Skilled labour is carried on with weak guiding impressions and weak consciousness.

It is more difficult to frame a diagrammatic representation of mechanism to fit our idea of synthesis, association, summation, inhibition. That which I am about to give is planned on a three-graded dichotomous type, and connotes what by many physiologists will be considered a false notion, viz., the representation of *different* peripheral parts in the *same* highest centre cell. Again, it assumes what anatomically we have no right to assume, viz., three levels of representative cells; all that we positively know as regards an anatomical

[1] For the sake of simplicity I leave out of view the anti-motor or inhibitory impulse.

justification of the schema, is that the total number of fibres between cortical and spinal cells is smaller than the total number of fibres between spinal cells and periphery.[1] Nevertheless, I have adhered to a three-level scheme as being most in accord with current psychological doctrine. And the representation of different parts in the same highest cell is in my view a material form of the psychological doctrine of the "unity of consciousness."

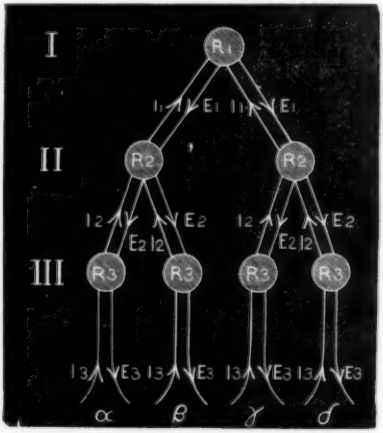


FIG. 2.

We cannot indeed make any sort of analysis of the centripetal process from impression to perception without imagining some sort of grading of nerve-cells; and the real anatomy of a hierarchic scheme is from this point of view quite subordinate to the ideal conception. The three terms adverted to above, viz., impression, sensation, perception, are as convenient and distinctive as any, and there is indeed no absolute necessity for adopting the further term apperception, which to some minds seems to possess very

[1] Sherrington's recent observation of "geminal" fibres in the spinal cord is perhaps another anatomical sign of an increasing number of fibres from above downwards above the spinal relay.

misleading connotations. The phenomena of hypnosis—more especially as presented to us in Bernheim's analysis—imperatively require us to adopt some such schematisation; *e.g.*, a hysteric hemianæsthetic, who asserts truthfully that she (or perhaps he) cannot feel, see, smell, taste or hear any impressions made on half her (or his) body, can be proved to have sensations of touch, sight, &c. He believes he cannot feel, and, therefore, does not feel, yet, all fraud excluded, it may be *proved* by appropriate devices that he does feel these sensations, but thinks that he does not do so, and, therefore, does not perceive them. Words are inadequate shortly to describe such a condition, yet it is an indubitable reality, and intelligible enough when regarded in terms of imaginary imprints on two different cerebral levels I and II. In the hysterical hemianæsthetic the imprint on II is effected, but the consequent imprint on I is travestied or untranslated owing to some functional disorder or incapacity of I, yet the physiological sequence from III to II and from II to I proceeds, and can be proved to proceed, if by any suitable device the false belief be circumvented—*e.g.*, such a person "blind" of one eye will see a real object doubled by a prism held in front of the sound eye; which proves that his "blind" eye is not "blind," that he has sensation by that eye when he does not think he is using it, but no sensation when he does think he is using it. Such a person may be and often is an impostor, but even a genuine sufferer would be set down as a malingerer in nine cases out of ten. Yet such a case might be perfectly genuine, the patient being at the same time "truly blind" of one eye, yet "truly not blind" of that eye, according to the sense in which we read these words "truly" and "blind." And the same may hold for any other sense; a genuine hysteric might be unable to hear or smell or move, because of her own conviction of impotence, yet able to hear or smell or move as soon as that conviction had been overcome. All these facts are best realised by a reference to two levels of cortical action II *sensory*, and I *perceptive*.

Although I shall refrain from entering upon a full and detailed consideration of the localisation doctrine, I can hardly

escape making some allusion to the obvious bearing of this upon the physiological doctrine of cortical representation, at the two opposite poles of which we have the "positive" contention of Munk, who experimentally verifies the projection doctrine of Meynert, and the "negative" contention of Goltz, who experimentally verifies the indifference doctrine of Flourens.[1] The antithesis thus shortly stated is at any rate as regards Goltz somewhat too absolute. Goltz indeed does not uphold absolute indifference, but rather an indeterminate and rudimentary predominance on a large scale and in the rough, of different function in different regions. His observations on dogs are irresistibly convincing; the observations of Munk upon dogs in proof of the projection of retinal upon cortical areæ, require stronger confirmation than they have yet received before they can be admitted as real; Schäfer operating on monkeys confirms the principle in a very guarded manner, and describes the projection very differently from the view of it given by Munk on the same animal. In the presence of such mutually destructive data, we are at liberty—pending further developments—to attach weight to purely psychological considerations. In accordance with the doctrine of the unity of consciousness, we are called upon to believe that our perception of things seen, things heard, things felt, &c., may take place in connection with the modes of motion of a single organ.[2] Pictorial and verbal expression of this theory or belief is given by Wundt in his doctrine of a frontal organ of apperception; the views of Hughlings-Jackson that the frontal cortex is possibly the highest level centre, and of

[1] Many other names might of course be quoted; Ferrier, Schäfer, Horsley and Beevor, Charcot, Déjerine, and most clinical observers are "localisers" more or less strict. Brown-Séquard, Schiff, Dupuy, are "indifferentists" with more or less reserve. But there are many shades and degrees of opinion between the two extremes. Schäfer, for instance, is in some respects (G. Fornicatus) a localiser, but in others (hearing) an indifferentist; Exner is a "diffuse localiser;" even Goltz may be characterised a "diffuse localiser" inasmuch as he makes reservations to the complete indifferentism of Flourens; and the position held by Brown-Séquard and by Dupuy is very similar.

[2] "We see that a perception can have in a nerve-centre no definite localisation, but only a diffuse localisation. No one excited fibre or cell produces consciousness of an external object; the consciousness of such external object implies excitement of a plexus of fibres and cells" (Spencer's "Principles of Psychology," second edition, 1870, p. 562).

Ferrier,[1] that it is connected with the intellectual faculties, are in perfect agreement with this well-known Wundtian schema. Considering the state of probability constituted by physiological experiments and by psychological considerations, I find sufficient justification for adopting a neural schema symbolising a superposition of heterogeneous impressions and a common origin of heterogeneous expressions $\alpha\ \beta\ \gamma\ \delta$, in and from a single highest level cell I.[2] Considering, however, the probabilities as regards the cortical mantle formed of many such connected cells I., and the subordinate masses of cells II. and III. I adopt a view intermediate between the positive pole "localisation" and the negative

[1] Accurately speaking, this view is unreservedly adopted by Ferrier in the first edition only of his "Functions of the Brain," although it still remains in his second edition. But quotations will best serve to show the subordinate importance and indeterminate character of the anatomical localisation as compared with the ideal conception. Ferrier's present view is substantially identical with that of Munk, although Ferrier very justly in another place (p. 429) criticises and rejects Munk's nominal antithesis, "psychical" and "cortical." But, as has been remarked, Munk uses the terms in a naked denotative sense, meaning no more than might have been indicated by the terms α and β.

"The centres of inhibition being thus the essential factor of attention, constitute the organic basis of all the higher intellectual faculties. And in proportion to their development we should expect a corresponding intellectual power.

"In proportion to the development of the faculty of attention are the intellectual and reflective powers manifested. This is in accordance with the anatomical development of the frontal lobes of the brain, and we have various experimental and pathological data for localising in these the centres of inhibition the physiological substrata of this psychological faculty" (Ferrier, first edition, p. 287.)

"The hypothesis of Hughlings-Jackson and others, that 'in addition to the sensory and motor substrata which have been demonstrated and defined by physiological and clinical research, there are other and higher motor as well as sensory centres, in which all the motor and sensory functions are again represented, and form the substrata of the higher mental operations . . . receives no confirmation from the facts of experiment, nor does it appear to me at all necessary to explain the facts of normal or abnormal mentation. We have in the sensory and motor centres of the cortex the substrata of the respective forms of sensory perception and ideation, and of the individual acts of volition, simple and compound, as well as of the feelings associated with their activity. It seems more reasonable to suppose that there may be higher and lower degrees of complexity or evolution in the same centres than to assume the separate existence of more highly evolved centres, for which no evidence is obtained by the results of experimental research'" (Ferrier, second edition, p. 460).

Wundt rejects the view that our supreme perceptions of different sensations are separately consummated in separate centres, and characterises it as a revival of the phrenological faculty scheme. (*Phil. St.*, vi., p. 1.)

[2] It is, perhaps, hardly necessary to say that the expression "a single cell I." made use of on account of the diagram, must be taken to mean "a single mass of cells I."

pole "indifference." I do not think that I can express that position either more accurately or more shortly than I have already done elsewhere.

"The 'nerve-centre' of mammalia and of man is a collection f nerve-centres occupying the cerebro-spinal axis, with more or less diverse special offices under their control—communicating each with the other upon occasions, yet separately active upon other occasions—having functions which are localised at certain parts, yet not strictly confined to these parts—playing upon and influencing each other in all directions, yet in some directions rather than in others, and maintaining some kind of precedence and rank, so that while all may influence all, yet some are usually guided and controlled by others—variously organised through past excitations, yet still variously organisable by excitations to come.

"The state of doctrine [*i.e.*, of localisation], may be made plain by an analogy. The various activities making up the business of the brain do not all take place all over its surface, as in a country without towns or villages, where all kinds of industry go on in every hut or tent; nor are the different activities absolutely restricted to certain spots, as if in walled towns. The brain cortex is not comparable with either of these extreme cases; its territory must be recognised as possessing towns with special industries, but towns with straggling and overlapping suburbs, and industries which are indeed predominant, each in a given centre, but not exclusive of all other industries in that centre, nor excluded from other centres in which other industries predominate."[1]

[1] My reading of Mosso's psychophysical observations (*Du Bois-Reymond's Archiv.*, 1890, p. 129), to the effect that prolonged intellectual effort subtracts from voluntary muscular workability, is in accordance with this intermediate view. If there are concentrations of different functions within an imperfectly differentiated organ, we should expect high-level exhaustion of any particular kind of activity to be greatest as regards that kind, but to exist in a minor degree as regards all other kinds of high-level activity. This, of course, applies only to the *central* component of fatigue, and not to the peripheral component attributable to the circulation of disintegration products. We may picture the functional depression as being of the entire cortex, but deepest at particular situations of concentrated activity. The converse change, viz., diminished capacity for intellectual effort after prolonged muscular exertion, is familiar to most brain-workers. On the other hand, most people have experienced that although one kind of mental activity fatigues the brain with respect to a different kind, yet such collateral fatigue is apt to be less marked than the principal fatigue, and so much so that a change of activity may feel restful.

And now, comparing the degree to which the functional and structural organisation has reached at different levels, I think we are justified in stating the following antithesis:

At the lowest level the representation is most definite and fixed, the canalisation of conductors is most closed, the differentiation of the central organ is most complete. At the highest level the representation is least definite and most fluctuating, the canalisation of conductors is most open, the differentiation of the central organ is least complete.

Contrasting the two extremes of cerebro-spinal action, we have in the purest reflex action an immediate, "fatal," unchosen response to peripheral stimulation; the nervous path is deeply canalised, alternative reactions are few, the particular reaction to a particular stimulus is easily predicted. In the purest voluntary action we have a delayed, "free," chosen response to a given sensation. The nervous path is not preformed; alternative reactions are numerous; the particular reaction to a particular sensation cannot be predicted.

It is another and a further and an essential question whether the greater diversity of function and division of labour is of such degree in the human brain, that particular parts of the cortex have become the special organs of particular parts and functions of the body. And towards the answer to this question of degree, the deduction from experiments upon animals is of little value; brain-surgery and clinico-pathological investigations of pure and uncomplicated cases of Jacksonian epilepsy and cortical monoplegia are the necessary and alone trustworthy premises of inference.

The localised effects of localised excitation of the cerebral cortex of dogs and of monkeys do indeed give fairly concordant results, which amply suffice to establish the principle that excitation of different parts of cortex and of subjacent corona gives different effects. But the converse test by localised extirpation has altogether failed to come up to the expectations that were first formed; in the hands of Goltz extirpations have been made of larger and larger areæ of the dog's cortex, of an entire hemisphere, and finally of both hemispheres, without producing permanent paralysis, while

in Munk's hands local extirpation of different areæ on the same kind of animal (not to speak of monkeys) gave different local and restricted loss of sensation and of motion.

As is well recognised, the contradiction does not merely illustrate that different observers looking for different effects *see* different effects, but is in part owing to whether immediate and temporary defects or permanent deficit (Ausfallserscheinungen) were described as paralytic effects. Although *a priori*, a temporary paralytic effect is a possible result of a cortical ablation, we have no right to assume *a posteriori* that the defect is due to lost central matter; it may equally well be an inhibitory effect due to irritated central matter, as has been urged by Brown-Séquard and by Goltz.

And it is one of the fundamental principles of the Nancy school that the symptoms of an organic lesion include not only such as may be termed direct and organic, the immediate result of destroyed tissue, but also a varying and variable swarm of satellite symptoms, the secondary and functional effects of the primary. The organic blot is, so to speak, surrounded by a functional halo; and it is one of our greatest sources of doubt and difficulty that these fringe effects may, to use the language of Brown-Séquard, be effects of dynamogeny or of inhibition, and that we may even with extreme care be totally unable to distinguish and assign to their separate cause (1) the organic paralytic effects, and (2) the functional paralytic effects, the ausfallserscheinung from the hemmungserscheinung, total loss from functional suppression. It would therefore be most unsafe to reject Goltz's contention that a true localisation of function requires as its proof a permanent and not merely a temporary defect. And yet a doubt remains, even the lapse of time yields no sharp line of distinction; when shall we say "this is finally lost, this is at present suppressed," after Bernheim has taught us to recognise in human pathology that widespread and *chronic* functional modifications surround the organic lesion and its direct physical effects. Every physiologist recognises the physiological importance of the distinction urged by Goltz; some have also recog-

nised the meaning of Brown-Séquard's language, but for the present it must, I fear, be admitted that very few of us sufficiently recognise how that fundamental conception underlies the curative treatment of the Nancy school. The medical (and human) aspect in this department is the department of the pure physician, the physiological (and human) aspects concern the pure physiologist.

The principle of functional diffusion from an organic focus, foreshadowed in the clinical study of Jacksonian epilepsy, clearly evidenced in the comparatively simple but scanty data of the laboratory, comes to its full maturity in the hardly less simple but far more numerous and more minutely readable data insulated for us in the Nancy doctrine of so-called hypnotism. As "true localisers" we should expect to find a limb completely paralysed after its cortical centre had been destroyed, *e.g.*, in a dog. But this is not the case; the limb can be moved, and at first sight its movements seem perfect; it is certainly not completely paralysed. On closer examination some defect of movement may indeed be detected, some clumsiness of the limb indicating that its movements are inaccurately co-ordinated, and these imperfect movements will probably be found to be such as are normally most highly specialised and *not* such as are most profoundly automatic. But even this amount of defect is not apt to be permanent—the apparent awkwardness soon wears off, movements seem to be relearned. The clinical history of a dog or of a monkey having suffered a removal of some portion of the Rolandic area altogether negatives a strict localisation of function, and at most suggests its local concentration. We may imagine a company of cells more particularly concerned in particular manœuvres of particular parts, and we see these manœuvres rendered clumsy by disturbance of the company; we are obliged to imagine that even if these cells are wholly destroyed, the lost control may become more and more perfectly assumed by other cells educated from the periphery, as the clumsied movements are repractised, corrected, and learned. We thus picture the cortical organ in a semi-fluid state of differentiation, still variable by new instruction, rather than

as a petrified and invariable collection of specialist organs tied down to particular functions and exclusively performing these functions.

Such diversity of duty gradually superimposed upon uniformity of capacity, seems to me a sufficient physiological ground upon which to reconcile the assured results of cortical excitation, the hardly less assured absence of permanent paralysis after cortical extirpation in dogs, and the psychological doctrine of the unity of consciousness in man. The view is (I think) substantially that offered by Wundt in his doctrine of apperception, and whether we go on to hold with Wundt that the seat of supreme or maximum consciousness consists in a frontal organ of apperception, or with Munk that it is the central area of each particular Fühlsphäre, it seems to me that we must admit the cortex to be *one* organ, *qua* the ideas and concepts composed by impressions confluent through different afferent channels. Wundt's view, to my mind, gives clear diagrammatic expression to this necessity, Munk's view leads us into the admission of departmental consciousness; the first postulates one supreme consciousness, the second postulates many supreme kinds of consciousness. Although we may (and must) admit a physiological division of function and of organ up to a certain point, I think that in the present state of knowledge we need not (and therefore must not) admit any psychological subdivision of the supreme organ of consciousness.[1] To take a very simple case, we must admit that the notion of a bell is given to us in *one* auditory-tactile-visual organ, even though there be a discrete centralisation of auditory, tactile, and visual impressions. Nor does it seem to me to be of essential importance whether we admit that supreme "composite" consciousness is the attribute of a part of the cortex, or of the whole cortex acting as one psychological organ of confluence although composed of differing physiological departments. What does seem to me to be of essential importance is that a physiological division of function, ascer-

[1] The phenomena of "audition colorée" (viz., the association of certain colours with certain sounds, the latter "suggesting" the former) naturally come to mind in this connection.

tained by experiment, should not lead us to admit a plurality of supreme consciousness.[1]

Guided by these considerations we may, I think, safely advance a step further in the *a priori* projection of thought:

The higher the brain the greater the mass of unformed formable material. But the further the differentiation shall proceed of formable material, the further it must already have proceeded of formed material. The higher brain must include subordinate highly specialised and economically-working mechanism-guiding parts. Otherwise it would not be free to be developed by new impressions and new combinations of impressions. And yet the further the brain has developed within the span of life of its possessor, the greater the differentiation of function and of organ out of the mass of formable material. We should expect greater concentration and fixity of function in the older brain, we find evidence of that greater concentration (with correspondingly dwarfed signs of dissimilar capacities) in the conduct of every matured and specialised man; experimenting on the cortex cerebri we should *a priori* anticipate clearer and sharper results from an old dog or an old monkey than from a young dog or a young monkey.[2] Thus it may be truly said, with reference to the functional attributes of the cerebral cortex that the higher the brain the less the fixity, the older the brain the greater the fixity.[3]

[1] Wundt has expressed this with great clearness in his recent criticism of Munk ("Philosophische Studien," vol. vi., 1890).

[2] Küssmaul makes the following apposite remark:—"One thing may be safely stated, however, and that is that the *age* of the patient is an important factor for the compensation of the injury. Children have been known to learn to speak after astonishingly extensive destruction of the left region of speech, and even of the entire cortex of the left hemisphere, while in old persons remarkably small foci of disease in the cerebral substance sometimes cause permanent aphasia." (Ziemssen's "Cyclopedia," vol. xiv., p.803.)

[3] The following quotation from Darwin ("Descent of Man," i. p. 38.)—"Little is known about the functions of the brain, but we can perceive that as the intellectual powers become highly developed, the various parts of the brain must be connected by the most intricate channels of inter-communication; and as a consequence each separate part would perhaps tend to become less well fitted to answer in a definite and uniform, that is, instinctive manner, to particular sensations or associations"—given by Dupuy as an opinion in support of the doctrine—"greater freedom of communication and of function in the higher brain," appears to me misleading in so far as the order of development is represented as being from the more special towards the more general, from heterogeneous towards homogeneous. The main drift of development exhibits itself as being towards specialisation, from the undifferen-

The schema given above (fig. 2) has, for several years past, furnished me with a concrete mechanical image to which I could refer the psychological phenomena of association.

If impressions of the two kinds α and β occurred simultaneously, and have been compounded in II., the repetition of α will be specifically liable to revive that compound effect which subjectively is the memory of α and β.

Similarly in the case of any other two or more kinds α, β, γ, δ, if their simultaneous or successive association in the past has been sufficiently frequent to leave a fused effect, the repetition of a single member α or β or γ or δ may revive that effect, *i.e.*, subjectively the memory of previously associated impressions.[1]

The same schema (fig. 2) has also served to materialise my conception of the influence of attention upon particular incoming or outgoing impulses. I imagine an effort of focalised attention to consist in a state of increased tension along any single tract, α or β or γ or δ, together with a state of diminished tension along the remaining tracts $\beta\gamma\delta$ or $\alpha\gamma\delta$ or $\alpha\beta\delta$ or $\alpha\beta\gamma$. In which conception I tacitly assume the senso-motor character of the series of

tiated towards the differentiated; still it must be admitted as conceivable that secondary connections between more or less differentiated centres may become established, making *against* rather than *for* their functional severance; but the main and primary movement is disconnection, reconnection is secondary and subsidiary.

Herbert Spencer in the 1870 edition of his "Principles of Psychology." (and also in the 1855 edition), gives the clearest possible *a priori* account of the localisation question. Alluding to the phrenological scheme of localisation, he says: "Whoever calmly considers the question cannot long resist the conviction that different parts of the cerebrum must, *in some way or other*, subserve different kinds of mental action. Localisation of function is the law of all organisation whatever; and it would be marvellous were there here an exception."

"Either there is some arrangement, some organisation in the cerebrum, or there is none. If there is no organisation the cerebrum is a chaotic mass of fibres, incapable of performing any orderly action. If there is some organisation it must consist in that same "physiological division of labour" in which all organisation consists; and there is no division of labour, physiological or other, but what involves the concentration of special kinds of activity in special places."

"Among fundamental objections to this view, *i e.*, the phrenologists," the first to be set down is that they are unwarranted in assuming *precise* demarcations of the faculties." ("Principles of Psychology," 2nd ed., vol. i., p. 573-4.)

[1] The Greek letters are not here used to denote subjective as contrasted with objective states.

cells I., II., III., and their susceptibility of undergoing augmentation or diminution of tension by impulses, *i.e.*, of undergoing alterations of excitability.[1] It is with this schema in mind that I am led to say, in somewhat figurative language, that "sensificatory stimuli affect us best when we meet them half-way by some action of our own, by an attitude of attention or by actual movements." In using such language I imagine a particular state of motion in I. causing in II. or in III. a state of increased excitability with regard to impressions homogeneous with previous impressions, of which the particular state of motion in I. is the material legacy.

§ 5—The Sense of Effort.

I have on a previous occasion insisted upon the close connection between the family of sensations, individually denoted as sensations of movement, sensations of muscular contraction, muscular sense, sense of effort, and their after-effect, the sensation of fatigue. From the physiological point of departure the phenomena of effort open to objective study are neuro-muscular phenomena; but from the psychological standpoint the phenomena of effort extend over the far wider area of psychical experience, and are mainly, if not exclusively, approached by subjective study. I have said mainly, because, although in many cases the psychological presentation has been exclusively introspective and interpretative, yet in others it has included certain objective results of deliberate experiment—to give an instance—the psychical experience denominated "effort of attention" has been introspectively analysed, but what may be termed the phenomenal fringe of the subject, has been experimentally studied by psycophysical methods.

It was objected to my presentation of the matter that the general term effort was misused. Although I had not

[1] Everyone will naturally call to mind in this connection thought-reading tricks, and actual alterations of nutrition, brought about by the excessively focalised attention, which is the essential feature of the hypnotic state; the observations of Tarchanoff (*Pflüger's Archiv.* vol. xlvi., p. 46) of local cutaneous electro-motive changes caused by attending to particular parts of the periphery, are also a physiological case in point.

specifically in view these forms of effort, I was alive to this use of the term; but I did not think it suitable to attempt to include them in the argument and I expressly limited that argument to the case of muscular effort. But so far from admitting that these more psychological forms of effort are to be dissociated from the most physiological form of effort, viz., muscular effort, I am disposed to insist upon the essential similarity of mechanism which runs through the whole range of effort experiences—sense of muscular effort, efforts of attention, of memory, of imagination, of will, of restraint. I can, however, do little more than offer an argument and state an opinion, appealing by the way to some few "experiments" (if experiments they may be called) which seem to indicate that an "effort of attention" is entitled to be spoken of as "nascent motor impulse."

We may attend to something we are *feeling*, or to something we are *doing*, *i.e.*, we may apparently have a *sensory* or a *motor* or a mixed form of attention, this, however, is no valid distinction, for in attending to what we are *doing* it is evident that we are attending to the thing being done and felt as well as to the thing about to be done. So far then the phenomenon of attention is consequent upon sensificatory antecedents.

But it is not possible to have any great concentration of attention without some alteration of motivity—movement or arrest of movement—greater muscular tension or smaller muscular tension—and the so-called thought-readers illustrate this fact clearly enough. In attending to what we are feeling we always do something, *i.e.*, use certain muscles. The phenomenon of attention is an antecedent of motor consequences. A desire (*i.e.*, an imagined effect, *i.e.*, a remembered similar effect) is the antecedent and the consequent of movement. The psychological—I had almost said dialectical—dilemma, as to whether attention belongs entirely to motor neural processes or whether it is merely a form of sensation, does not seem to me a very serious one in terms of psychical phenomena.

Attention is "increased sensomotility," at the same time

consequence of sensificatory antecedents, and antecedent of motor consequences. I use this word to denote that *one* property of a higher nerve-centre which has been improperly chopped into two properties, sensibility and motility of sensory and of motor centres. It is the homologue of the most general term excitability and of the most special term suggestibility. "Excitability," a name for the property of certain centres, motion with sensation. "Suggestibility," a name on the same pattern, from suggestion to conduct.

Let us review some cases to test the applicability of our definition—attention increased sensomotility. A chef d'orchestre picking out a weak instrument, a schoolmaster keeping an eye on a particular boy, a meditating monk or a howling dervish, a student in presence of a subject to be mastered, in short anybody doing anything, has his attention focalised upon his task in hand; we say that he concentrates his attention by voluntary effort; but if we imply that he originates the effort of will, we go beyond our tether.[1]

It is most in accord with the principle "voluntary action

[1] I do not clearly understand Bastian's distinction ("On the neural processes underlying attention and volition") between the neural processes of attention and of volition, nor his conception of voluntary attention as "a compound or fusion of the two states volition and attention." The *two* names seem to me to denote *one* state or process or "alteration of sensomotility;" we have always diffuse sensomotility (states of attention and of inattention) and the plus state of sensomotility or "ad-tentive" state may explode in one volitional act or confine the explosion of other volitional acts; attention is nascent volition, volition is expressed attention; or otherwise, thought is nascent action. I cannot call to mind any example of purely sensory attention without motor concomitants; the indivisible double aspect of the process imposes itself upon me with such force that I am obliged to think of it under the compound word alteration of sensomotility. Not implying, however, that the process is to be regarded as compounded of sensory and motor factors, but to indicate that it exhibits subjective with objective symptoms—alterations of feeling with alterations of attitude—although itself a single and indivisible phenomenon. To the objection that we may have altered attention without altered movement, I would reply that such an apparent instance of pure sensory attention is a fallacy, and that an unseen motor consequence can always be made manifest as a visible motor consequence by intensification of attention without alteration of kind of attention.

To another possible objection that attention may have for its consequence not motion but arrest of motion, it is enough to reply that arrest of motion is as much a motor phenomenon as motion itself. If I attend to an object at the periphery of the field of vision, the cerebral process includes the production of an intensified fixation of the point of regard. If by an effort of imagination I reverse the position or relief of a transparent vessel, the cerebral process includes the production of an alteration in the diameter of

a disguised reflex" to refer attention, the psychological phenomenon, to increased sensomotivity, the physiological phenomenon. We can often enough in given cases discern the antecedent stimuli that may have elicited states of attention, but when we attempt to get behind the phenomena at the true cause of the state, we recognise that we are in presence of an ultimate property of cerebral living matter. We do not know.

We have, however, in the conditions and consequences of variations of attention, a field of exploration extensive enough to satisfy the most industrious aspirations to positive knowledge. Hypnotic phenomena, education, mental health and mental diet, in a word the influence of man upon man, are in the main determined through varying attention, varying sensomotility, varying influences by varying influences. And as a right theory is often the motive influence of right practice, so it becomes of great importance to acquire a right view of the physiological determinism of the phenomena of attention.

Much of our most valuable knowledge is analogical, one of the most valuable analogies with reference to attention, is afforded by retinal phenomena; the analogy is more than an analogy, for the retinal phenomena that we have to utilise are in reality retino-cerebral, so that a study of retinal

the pupil. Every antecedent has its consequences, every consequence has its antecedents, any phenomenon is a transverse section in the stream of change. Every centripetal impression has its motor consequences, every movement has its centripetal antecedents, any central neural phenomenon is a transverse section in the stream of neural change. We may call the stream past and future, centripetal and centrifugal, but the phenomenon itself is both or neither. I conceive the physiological phenomena underlying "emotion" as being similar in composition to those underlying attention, *i.e.*, as sensomotile, and not purely sensory nor purely motor, nor a resultant of separable sensory and motor factors. We know subjectively that an emotion entails feeling as a resultant of centripetal impulses, objectively that it entails movement or arrest of movement, the resultant of centrifugal impulses. Emotions are feelings expressed by motions, and the motor element is even more obviously manifested in emotion than it is in attention. But of the process itself we cannot say in either case that it is either *only* sensory or *only* motor; objectively imagined an emotion is the result of an alteration of nerve-substance, which alteration is the consequence of centripetal impulses and the antecedent of centrifugal impulses. And it is no drawback to the second half of this view, that the expression of an emotion can be restrained, for in restrained emotions there is the centrifugal tension or tendency, and it may be, actual movements suppressive of a natural muscular expression.

focalisation and of retinal contrast is in large degree a study of cerebral focalisation and cerebral contrast, which are among the most important phenomena of attention. I am aiming at one particular group of considerations that can, I think, with advantage, be presented from a very general point of view in this connection, viz., contrast phenomena.

Stated in general terms:—the value of any given constant is estimated to be + or − according as its surroundings are − or +; magnitudes are over or underestimated amid smaller or larger surrounding magnitudes, colours are intensified or weakened according as they are observed on similar or upon complementary[1] backgrounds; and psychologically any sensation is intensified or weakened according as it occurs upon a similar or a dissimilar field of attention. This law of contrast, simultaneous as well as successive, applies to the two portions of the field of attention which we have referred to as "distinct" and "indistinct"; and here again we may, with profit, represent by a ratio $\frac{\alpha}{\beta}$ the sensation resultant from its two component sensations α and β, α being the distinct area and β the indistinct area of attention. This resultant sensation will obviously vary with greater or smaller concentration of attention, according as the distinct component α forms a larger or smaller proportion of the total α and β. And now the law of contrast is evidently applicable; just as two antagonistic colour sensations mutually intensify each other, so do any two antagonistic masses of sensation; in this case the antagonists are the more intense distinct and the less intense indistinct portions of the total sensory field; the first diminishes the second, the second diminishes the first; in the hypnotic state the first annuls the second, and attention is at its maximum of concentration. Fixed attention is in fact the essential element of hypnosis of all degrees and however produced. In the normal state a person may be more awake to a particular kind of stimuli, less awake to all others; he is "pre-occupied," and his pre-occupation may be trifling or moderate or considerable. In the hypnotic state it is excessive, the hypnotised person is over-sensitive to a

[1] Or antagonistic (Hering).

certain focalised portion of his environment, under-sensitive, or it may be impervious towards the whole unfocalized field.

In my previous paper (*Sense of Effort*, BRAIN, 1891), I offered as additional to the exclusively cerebral or spinal reflex view of movement, the following groups of considerations.

The initiation of action — Hitzig's image of movement, Munk's Gefühlvorstellung or Bewegungsvorstellung, Ferrier's motor idea or impulse, Bastian's revival of kinæsthesis—is demonstrably attended with a consumption of force-producing material.[1] With this change manifested as diminished workability (motor fatigue) we have a sensation popularly termed fatigue (sensory fatigue), and I have urged above that the association of the two aspects is such as to forbid us to dissociate them; "motor" fatigue and "sensory" fatigue are one and the same thing, to which for want of a better word we may apply the term senso-motor fatigue. Moreover I have argued in my previous paper that the incidence of "fatigue" (which is the *result* of movement) may be taken as a guide to the incidence of "effort" (which is the *concomitant* of movement), and have concluded that "effort" and "fatigue" depend upon *changes at the centre and at the periphery*.[1]

[1] This argument was the main motive of my paper. I find that James, who ostensibly pronounces himself in favour of the entirely peripheral origin of effort-sensation, commits himself to the position I take, viz., the distinction between peripheral and central factors.

"Effort of attention is thus the essential phenomenon of will. This volitional effort, pure and simple, must be carefully distinguished from the muscular effort with which it is usually confounded. The latter consists of all those peripheral feelings to which a muscular exertion may give rise ("Psychology," vol. ii. p. 562.)

Dr. Delabarre ("Mind," July, 1892, p. 383) objects that the fault of my reasoning lies in its major premise, which contains three unwarranted assumptions: (1) That the objective signs of exhaustion are always indicative of a previous expenditure of energy in the same parts. (2) That a subjective sense of fatigue is indicative of a corresponding previous effort and sense of effort in the same parts. (3) That objective signs of exhaustion are indicative of a subjective sense of fatigue, and objective signs of effort of a subjective sense of effort, localised in the same parts.

I really cannot discern these three assumptions in my major premise, which was clearly stated and printed as follows: *The material changes subsequent to action, are co-extensive in the motor organ with the material*

I admitted the peripheral sensificatory factor as matter of common knowledge, but offered evidence against the view that it is the sole factor of the sensations of motion to the exclusion of the concomitants of central emission. I also admitted as matter of common knowledge

changes accompanying action. The three assumptions are introduced by Dr. Delabarre rather than implied by me, and his objections to them are not objections to the premise itself.

This is a sufficient answer to the criticism, but I wish to clear the issue rather than to retrench myself behind a logical defence, and will therefore reply as follows to Dr. Delabarre's three assumptions:

(1) That Mosso's experiments (alluded to on p. 368 of this paper) do not appear to me to be at variance with the general truism that effects and after effects must have the same loci of incidence. The truism applies to the area of diffusion as well as to the focus of maximum at which the expenditure of force-producing material and the production of fatigue-products is presumed to occur, and experiments do not allow us to hold that the fatigue-products thrown into circulation can ever produce an effect at all approaching in intensity the local effect at the place of origin of such fatigue-products. (2) Delabarre's objection that the second assumption "is negatived by the fact that the feeling of fatigue located in the eyelid is not at all commensurate with the amount of work this organ has accomplished, and is sometimes excessively strong in the morning, after complete repose of the muscles involved," introduces terms of comparison from which I carefully refrained, indicating as my reason that the introspective comparison of sensations is particularly liable to fallacy in this connection. I had and have nothing to say about any correspondence of magnitude between fatigue and effort, being well aware of the fact that the apparent magnitude of feeling varies with associated and contrasting circumstances. To trace any correspondence between two subjective magnitudes it is necessary to secure identity of circumstances and conditions. (3) The third assumption that objective signs of exhaustion may be utilised as indicators to a subjective sense of fatigue, although not contained in my major premise, is however made in the inference from premises to conclusion. So far from ignoring it and letting it smuggle itself unperceived into the argument, I repeatedly pointed out this assumption as the weak link in my chain of proof (Sense of Effort, p. 191, 192, 433). It was, so far as I could see, the one logical breach that might be employed by an adverse critic to break the proof, and I was careful myself to indicate that breach; I claimed no more than the *highly probable* parallelism between

$$\frac{\text{sense of effort}}{\text{kinetic effect}} \text{ and } \frac{\text{sense of fatigue}}{\text{loss of kinetic power}}$$

and therefore the highly probable character of the conclusion at the end of the inference. But I urge in defence of this breach three considerations: (1) That the weak link in my chain is no sign of the strength of any rival chain. (2) That "to take the demonstrable signs of material changes of objective physiological fatigue as indicators to the undemonstrable material changes of subjective psychological fatigue, keeps us closer to phenomena than the hitherto universally adopted procedure of drawing conclusions from the suggestions afforded by simple introspection." (3) That, although probability is not proof, yet proof is no more than overwhelming probability, so that in the opinion of some the high probability of my conclusion may be held as amounting to proof.

Dr. Delabarre himself grants as much, and rather more, in some introductory sentences that may be compressed as follows: "Granting that objective

that the various components of the sensificatory factor arising from skin, joints, tendons, and possibly muscle itself, must be perceived centrally through afferent channels. I see no reason for adopting the theory of Lewes, that our knowledge of motor change can be derived from centripetal impulses along the efferent tract.

signs of exhaustion are indicative of a subjective sense of fatigue, and granting that the exercise of central energy is indicative of a sense of effort localised where the effort takes place, the central sense of motor innervation is proven." And although it may be psychologically allowable to say that "we know without elaborate experiment that the central organ exerts energy," I, for my part, prefer to this *a priori* and indefinite premise, observed physiological data, *i.e.*, objective and measurable signs of central expenditure.

Professor G. E. Müller (*Zeitsch. f. Psychologie, u.s.w.*, 1892, p. 122), also adversely criticises my paper on the "Sense of Effort." The following sentence sums up his criticism:—"Verfasser schliesst auf die Entstehungsweise der normalen Empfindungen des Muskelsinnes ohne Berechtigung aus der Entstehungsweise der Ermüdungsempfindungen. Auf letztere schliesst er ohne Berechtigung aus der Entstehungsweise der objectiven Ermüdungserscheinungen, denen er ohne stichhaltige Beweisgründe einen vorwiegend zentralen Ursprung zuschreibt." That is to say, he rejects my data and my reasoning. As regards data, time and space do not allow me at present to review them in detail, but I shall take an early opportunity of doing so with reference to Prof. Müller's searching and therefore valuable criticism. I am well aware of their imperfection, and hope that other observers may assist in their experimental purification and prosecution. As regards reasoning I wish Prof. Müller had not gone outside the inference as formulated by me on p. 190. I urged a conditional or probable conclusion, and he represents me as claiming to have produced an unconditional proof. I had guarded myself from this claim as clearly as I was able, *e.g.*, on pp. 190, 191, 192, 433. I admit that the sequence considered by Prof. Müller to be "ohne Berechtigung," does fairly and correctly represent the view I held and hold, but I cannot accept the omission of the qualifications that I recognized to be necessary. The main object of my paper was to urge the examination of this complicated matter with the neuro-physical change (objective) rather than the psychical perception (subjective), as the point of departure. Whereas psychologically we have to deal with four (or more) distinguishable kinds of phenomena produced in voluntary movement, physiologically we have to deal with one kind of phenomenon, *i.e.*, the material (motificatory and sensificatory) change, and upon a balance of evidence and of probability to form an opinion as to the organic distribution of that change. Starting from the concept of a single neuro-physical change with kinesis as its objective expression, æsthesis as its subjective concomitant, I examine the kinetic power at the centre and at the periphery as the accessible objective indicator to the neuro-physical change producing kinesis and æsthesis. I do not see reason to admit that kinesis and æsthesis are produced by separate neuro-physical changes, still less to admit that qualitatively distinct æstheses in the same neural segment by the same cause (*e.g.*, sense of movement and sense of fatigue) are produced by separate neuro-physical changes.

The hypothesis serving as my guide may be put thus:—

I. Neuro-physical change occurs with voluntary motion, (1) in the cortex, (2) in the spinal cord, (3) in the end-plates, (4) in the muscle, (5) in the tendons, articulations and skin. 1 and 2 are central, 3, 4 and 5 are peripheral. [For reasons given in my previous paper (p. 181) I exclude nerve-fibres from consideration. Nor is it necessary in the present connection to consider a sixth component, viz., spinal and cortical

I do not share this view because I see no evidence to support it. Indeed, the supposition has appeared to me to be so purely gratuitous that I did not even mention it in my previous paper.

changes consequent upon 3, 4 and 5; these secondary central changes are admitted by every one, and to include them in the present field of thought might possibly distract attention from the point at issue, viz., the existence of primary central changes; whether such "primary" changes are truly primary, *i.e.*, uncaused, or resultant from antecedent centripetal impulses is another question that has already been considered in this paper.]

II. The sensation of motion arises from neuro-physical change in one, some, or all of these situations.

III. Neuro-physical change in consequence of voluntary motion, having as its objective sign physiological or "objective" fatigue, occurs in 1, 2, 3, 4 and 5.

IV. The sensation of fatigue arises from neuro-physical change in one, some, or all of these situations.

I find evidence (criticised and rejected by Müller) that objective fatigue is central and peripheral, I infer that the neuro-physical change, with motion and after motion, is central and peripheral. If the sensation of fatigue is the subjective concomitant of that neuro-physical change, then subjective fatigue has central and peripheral sensificatory factors. If the sensation of movement is the subjective concomitant of the neuro-physical change, then the sensation with movement has central and peripheral sensificatory factors.

As far as I can see, the acceptance or rejection of my reasoning from the phenomena of objective fatigue to the distribution of its neuro-physical basis, and from the latter to the distribution of the neuro-physical basis of objective movement, and of the sensations of fatigue and of movement, must in last resort depend upon a preference between the following conceivable alternatives. The objective movement and the subjective sensation of that movement depend upon the disintegration of *one* neuro-physical substance S. M. or of two distinct neuro-physical substances S and M. The two alternative con-

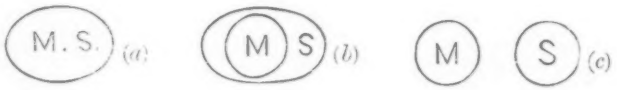


FIG. 3.

ceptions may be symbolised by the (*a*) and (*c*) in fig. 3. An intermediate alternative in which all neuro-physical matter is regarded as sensificatory, but only some such matter as also motor (some being only sensificatory) may be symbolised by (*b*) in fig. 3. I regard concept (*a*) as the most probable, concept (*c*) as the least probable. Concept (*b*) which represents that material other than inogenic may contribute to æsthesis, is in this connection nearer to alternative (*a*) than to alternative (*b*), if we recognise that every sensificatory change influences centrifugal tension. Subjective fatigue and the sensation of movement depend upon the disintegration of *one* neuro-physical substance or of *two* distinct neuro-physical substances. I regard the former as the more probable alternative. "*Neither more, nor more onerous, causes are to be assumed than are necessary to account for the phenomena.*"—(Hamilton.)

Dr. Bastian invokes Gotch and Horsley against it in the following terms:

"The lack of sensibility accompanying the action of motor centres has now been experimentally demonstrated by Gotch and Horsley."

I do not know whether Dr. Bastian has fully appreciated the gaps and pitfalls between Gotch and Horsley's data, and the conclusion which he draws from them. But in so far as he is a disbeliever in a retropulsive wave along an efferent tract he is, in my opinion, justified. The Bell-Majendie experiments on nerve-roots prove this point, by demonstrating that it is not possible (in the absence of recurrent sensory fibres) to obtain a reflex effect by stimulation of the central end of an anterior root, or by stimulation of a mixed nerve after section of the posterior roots.

Gotch and Horsley,[1] although they have not gone so far as to claim to have "experimentally demonstrated the lack of sensibility accompanying the action of motor centres," have indeed drawn a conclusion which does not seem to me to depend upon the data from which they draw them, but upon other and far more assured and familiar data. They consider that:

"As the electrical method affords the first opportunity of an experimental contribution to this subject, it is interesting to find how strongly its application bears out Bastian's position. In this chapter it appears probable that the kinetogenetic portion of the centre is the afferent [*i.e.*, that in which the potential energy is converted into kinetic] side of it, and the more especially when it is seen how readily the centre discharges into the afferent nerve channels (*i.e.*, actually 'backwards' as compared to the course of ordinary afferent impression)."

"The method has further enabled us to ascertain what connections and facilities for conduction the efferent, or so-called motor side of a nerve centre possesses, and instead of finding, as might have been expected from the ordinarily expressed beliefs on this subject, that we have to deal with a source of energy that was readily aroused, and freely connected with its neighbours, we found, to our surprise, that it afforded nothing of

[1] Croonian Lecture. *Phil. Trans. R.S.*, 1891. Summary of Cap. xi., p. 509).

the sort, and that its power of conducting impulses centripetally was practically *nil*.

"Curiously enough, this last point was foreseen also by James, who in his celebrated work on the 'Feeling of Effort,' 1880, while endorsing the views of Bastian, says that 'the electrodes of the physiologist,' if applied to the central end of the anterior root, would not arouse any 'sentient,' *i.e.*, afferent impulse in the cord. We are happy to find that our experimental results, unusual though they were, are, nevertheless, in close agreement with the deductions of the logical method of these distinguished writers."

I do not dispute either the electrical data, or the conclusion that impulses do not reach the brain from muscle by running up efferent tracts, but I do not think that the latter is borne upon the former; to my mind the facts indicate an altogether different kind of conclusion, and the conclusion is established from far more direct and simple data. As regards the second point, it is enough to remark that the absence of movement on stimulation of the central end of an anterior root—forming part of the ordinary class demonstration of Müller's experiment on the nerve roots of the frog, and of Majendie's classical demonstration of the same fact on mammalia—proves it conclusively. As regards the first point, while it is possible that the facts may be congruent with this established truth, it is equally possible that they may illustrate quite a different matter; it is well known that the negative variation in nerve is elicited in far greater amount by electrical stimuli of nerve than by either functional or mechanical stimulation;[1] doubt has been felt and expressed whether the "negative variation" and the nerve-impulse are co-extensive phenomena;[2] the large variations up and down the uninterrupted fibres of the posterior roots and columns, as compared with the small variations down the anterior roots and the absent variations up the cord

[1] Du Bois-Reymond. "Thierische Elektricität," vol. ii., chap. 6, p. 507. Electrical excitation gave a deflection of 40°, whereas non-electrical excitation gave only 1° to 3°, and the powerful functional tetanisation produced by strychnia gave 1° to 3° or even 4° deflection.

[2] Kuhne. "Zeitschr. f. Biol.," xxii., p. 339, 344 *et seq.*, in admitting "double conduction" in nerve, bases his conclusion upon the results of his gracilis experiment rather than upon the evidence by negative variation.

above the anterior cornual cells, suggest that they are dealing with an electrical phenomenon depending (at least in part) upon the physical continuity or discontinuity of fibres, rather than with any pure and simple electrical *measure* of the magnitude of functional disturbance in the nerve. That this last assumption (*i.e.*, measurement of functional impulse by its electrical indication, or properly speaking by galvanometric deviations) is improbable, is further indicated by some of the authors' own results. They obtained large effects down the afferent tract with centrifugal discharge, and diminished effects down the efferent root; in most cases this centrifugal discharge was by electrical excitation, but in a few cases it was functional discharge; these few cases are the real key to the validity or the reverse of Gotch and Horsley's contention that nerve-impulses pass freely down (as well as up) the afferent tract and root, diminished down the efferent root from the cord, and not at all up the efferent root to the cord. But even if the contention be admitted as valid, it is difficult to see what additional cogency as regards the kinæsthesis doctrine would be added to the far more cogent facts (1) as to the afferent root, the known failure of response by excitation of its central end, and (2) as to the posterior root, the known passage of centripetal impulses along it. The non-existence of an effective retropulsive wave *up* the efferent tract is proved by (1). The existence of a retropulsive wave *down* the afferent tract (as urged by Gotch and Horsley), interesting as it may be, would add no strength to our knowledge of the centripetal transmission of peripheral sensificatory stimuli.[1]

It is difficult to understand how any logical thinker can be led to translate this maze of inference into "an experimental demonstration of the lack of sensibility accompany-

[1] If more proof were needed of the non-existence of a retropulsive wave from muscle up motor nerve we might adduce the observations of Kühne, who altogether failed to find any evidence of its existence in experiments made *ad hoc* upon the frog's gracilis ("Zeitschr. f. Biologie," xxii. p. 333).

Du Bois-Reymond, discussing the evidence of double conduction afforded by the negative variation, remarks that the absence of reflex response to stimulation of the central and of a motor conductor is intelligible on the assumption that the central stations of motor nerves are inexcitable by the retropulsive wave ("Thierische Elektricität, ii., p. 574).

ing the action of motor centres." It will be very much to be regretted if this verbally clear but phenomenally obscure dictum should be uncritically quoted as a "datum experientiæ;" I have therefore done my best to disentangle the argument and to make plain its possibilities and probabilities of fallacy.

This has been a side issue; so far from having led into it, I deliberately ignored it in my first paper after the most careful consideration; but as it has been raised in authoritative quarters (in a presidential address to the Neurological Society, and in a Croonian Lecture to the Royal Society), it had to be entered upon.[1]

I have also advanced what I consider to be a very important extension of view with regard to the neuro-muscular government of the body, viz., that in addition to

[1] Bastian, until quite recently (BRAIN, 1887, p. 44) quoted Wundt and Bain as the exponents of the cerebral emission theory. I pointed out (BRAIN, 1891, p. 436) the injustice of attributing to Wundt an opinion expressly repudiated by him, and signified adhesion to Wundt's real opinion, as contrasted with Bain's. And Bastian now (BRAIN, 1892, p. 2) makes a similar distinction between Bain and Wundt. This is the passage upon which I had relied as regards Bain's position:—

"As the nerves supplied to the muscles are principally motor nerves, by which the muscular movements are stimulated from the brain and nerve-centres, our safest assumption is that the sensibility accompanying muscular movement coincides with the outgoing stream of nervous energy, and does not, as in the case of pure sensation, result from an influence passing inwards, by ingoing or sensitive nerves. It is known that filaments of this class are distributed to the muscular tissue, along with the motor filaments; and it is reasonable to suppose that by means of them the *organic* states of muscle affect the mind. It does not follow that the characteristic feeling of exerted force should arise by an inward transmission through the sensitive filaments; on the contrary, we are bound to presume that this is the concomitant of the outgoing current, by which the muscles are stimulated to act. No other hypothesis is adequate to represent the total opposition of nature between states of energy put forth, and states of passive stimulation."—Bain, "Senses and Intellect," 2nd ed., 1864, p. 92.

But I now learn from Prof. Bain that he also admits the participation of a peripheral factor.

So that, after all, it is permissible to bracket Bain and Wundt, not as maintaining the exclusively central source of the sense of effort, but as holding it to be of mixed origin; the two names need not be separated. I have hopes that it may eventually be allowable to add Bastian's name to these; for the present, however, this addition is not permissible, he still holds to the exclusively peripheral origin of the sensificatory changes with *muscular* effort, and distinguishes this form of effort as differing in kind from effort of attention, volition, &c., of which he presumably admits a *central* material substratum. Of course, by saying that Bastian holds to the exclusively peripheral origin, &c., I do not mean to imply that he ignores the truism that sensation is *consummated* centrally. But for the present he holds that the sensation of movement (kinæsthesis) is *initiated* solely at the periphery, whereas Wundt (and Bain) hold that it is initiated at the centre, and at the periphery.

(1) central initiation,[1] (2) reflex response, we must admit (3) direct response, in the perfect quantitative co-ordination of muscular movements.

This third item was very cursorily alluded to in my previous paper in a note to p. 244, and opportunity may therefore be taken to more adequately develop it.

The view that reflex spinal action plays a very large part in the government of muscular action is very generally accepted, and the extent to which spinal control is held to be effective has of late years been greatly augmented. Among published papers I may quote Exner's[2] and Chauveau's.[3] I may add that in 1880, when the question occupied my thoughts very largely, I was strongly inclined to the view that reflex muscular action responsive to the extension of muscle or tendon or ligament must be a chief factor in the co-ordination of muscular movements. I still admit this view, but not to the extent that I might have done, nor as unreservedly as is done by Exner, Chauveau and many others, owing (1) to the fact which I demonstrated in 1880,[4] and (2) to the important principle long ago discovered by Heidenhain,[5] but even now scarcely appreciated at its full value by neurologists or by physiologists.

(1) The positive item which withheld me from supposing that muscular co-ordination is altogether by reflex action from the cerebro-spinal axis, was the phenomenon of "tendon-reflex," which is demonstrably not a reflex from the cord, but a direct phenomenon of which a continuous reflex tonus is a necessary condition. The phenomenon being recognised as a direct response to an extensile excitation, we cannot fail to recognise the direct excitability of muscle itself by sudden tension, as well as its indirect excitability

[1] This word might be taken in an active or in a passive sense, to imply a spontaneous or a provoked initiation of nerve-impulses. It is here taken in the second sense in accordance with the physiological postulate: voluntary action = a species of reflex action. But see also the concluding paragraph of this paper.

[2] Exner. Ueber Sensomobilität. *Pflüger's Archiv.*, xlviii., 1891, p. 592.

[3] Chauveau. On the Sensorimotor Nerve-circuit of Muscles. Brain, 1891, p. 145.

[4] "On Muscular Spasms known as 'Tendon-Reflex.'" Brain, 1880, p. 179.

[5] Heidenhain. Mechanische Leistung, Wärmeentwickelung und Stoffumsatz bei der Muskelthätigkeit. *Leipsig*, 1864.

viâ a central controlling organ, as being contributory to a balance of power between muscles, variously stretched by each other's contraction, and as supplying a most rapid corrective of any sudden fault of balance.

And this direct responsibility being recognised, we cannot fail to see that all that is said in exposition of the reflex adjustments of position by reflex contraction responsive to tension, applies with additional force and even clearer "zweckmässigkeit" to direct adjustments of position by direct contraction responsive to tension.

The principle may be summed up in the short dictum—*ut tensio sic contractio.* As has been said the visible contraction, in response to a direct extensile stimulus, requires, as a necessary condition, that subsensible degree of reflex action, which, for want of a better name, is called reflex tonus, and if the reflex arc is cut at any point the direct contraction fails. But the dictum "*ut tensio sic contractio*" may be discovered to apply also in some degree to muscle isolated from the nervous system.

(2) The principle of muscular action contained in Heidenhain's often quoted but little known monograph, published nearly thirty years ago, may be summed up in this double proposition:—*ut tensio, sic labor, sic calor.*

The proof that muscular work increases with muscular tension is contained—I had almost written concealed—in every text-book of physiology. Up to a certain limit an excised frog's muscle does more work in contraction the greater the extending load, and within a somewhat narrower limit the actual contraction or shortening increases with the load. Thus we have illustrated the dictum "*ut tensio sic contractio*" in the accurate sense of the word contractio, *i.e.*, amount of shortening, as well as the dictum "*ut tensio sic labor.*" It is important to recognise now that in our first use of the dictum "*ut tensio sic contractio*" we have not used the word in its accurate sense but have included in it connotations which properly belong to the word "*labor.*" When we summed up the principle of direct response to extension in the words "*ut tensio sic contractio,*" this last word implied amount of work as well as degree of shortening in

the muscular contraction, and the proposition "*ut tensio sic labor*," which as we have seen applies to excised muscle, might therefore have been substituted for it.

Heidenhain's observations show further that the increased work with increased load does not occur at the expense of the other form of actual energy manifested by contracting muscle, viz., heat, but that with increased load there is increased heat as well as increased work. *Ut tensio sic calor* as well as *ut tensio sic labor*.

These relations teach us that, other conditions being constant, but with variable load or tension or resistance to contraction, the amount of energy manifested by isolated muscle is variable. The strength of contraction (*labor*) varies with the mechanical task to be accomplished; more weight or more tension or more resistance to contraction elicits a larger production of muscular energy in the forms of work and of heat, the isolated muscle evolves energy in proportion to demand by tension.

This is the second feature to which I have referred to above as the "direct responsibility" of muscle, a property which in my opinion plays an important part in the accomplishment of exact movements appropriate to varying states of tension.[1]

§ 6—Apperception.

We have already had occasion to refer to Wundt's theory of Apperception (p. 372). It is indeed impossible to make any progress in the analysis of cortical function from either a physiological or a psychological point of departure without

[1] The principle appears to me to be of such importance that we ought to be particularly careful not to admit it uncritically, but on the contrary to expressly recognise the limited extension of the heat-data that form its chief basis. Heidenhain's conclusion is exclusively based upon experiments with excised frog's muscle; attempts to verify the principle with mammalian muscle made in the Leipsig laboratory by Lukjanow (*du Bois-Reymond's Archiv.*, 1886, suppl. p. 117) yielded in this respect a perfectly negative result, the principal *ut tensio sic calor* could not be experimentally verified. On the other hand it is one of the principal conclusions of Chauveau's researches on human muscle (*Le travail musculaire*, Paris, 1891), but in this case a critic acquainted with the subject might easily find technical loopholes for objection. Another point of deficiency that can only be filled in by thermo-electric experiments of extreme delicacy is the failure of proof of any difference in amount of heat evolved by the extension of living *versus* dead muscle.

involving oneself in a consideration of that theory. The nature of the problems of which it is offered as an explanation, is such that the explanation itself requires much further explanation, and that it has been read or imagined as conveying a meaning diametrically opposed to the whole spirit and tendency of Wundt's teaching. No one has abjured faculty fetishism in psychology more plainly and explicitly than Wundt, yet we find him repeatedly pointed at as a faculty-monger and triumphantly refuted.

Of recent years this misrepresentation has been particularly noticeable in England, and judging from a review of Münsterberg's Beiträge by Dr. Titchener, the fashion seems to have been set in Germany.[1] Yet, making the most liberal allowances for the intrinsic difficulty of the subject, and for the natural preference by a hurried reader for second-hand allusions written in English to the original expositions written in German, the travesty remains an amazing one to anyone acquainted with Wundt's writings. To go back no further than the beginning of this year, we may refer to the last presidential address to the Neurological Society as an instance in point. Wundt's "so-called faculty of apperception" (p. 20) is there alluded to and refuted, but to the reader acquainted with Wundt's writings there is hardly any trace of Wundt in the so-called Wundtian position there presented and demolished. Indeed it is only too clear that this authoritative contradiction, which may not unreasonably be expected to influence opinion in this country, is based upon the case as presented by Wundt's adverse commentators rather than upon that contained in Wundt's own declarations, and the Wundtian position carried by Dr. Bastian seems to me to represent not what Wundt thinks, but (on p. 20) what Croom Robertson thinks Münsterberg thinks Wundt thinks, and (on p. 15) what Bain thinks Wundt thinks. The bald faculty puppet presented to us

[1] "Dr. Münsterberg's polemical aim is the demolition of a *soi-disant* 'theory' or 'metaphysic' of Apperception. Like pulpit atheism, this theory is a thing of straw, which the author sets up and knocks down with a wearisome monotony. The serious part of the matter is that he has fathered the 'metaphysic' upon Wundt" (*Mind*, 1891, p. 530.)

by Bain[1] and by Bastian as Wundt's child, was long ago torn to pieces by Wundt himself[2] (*Phys. Psych.* 1886, p. 354). This much is simple matter of historical fact to be verified or refuted by reference to Wundt's classical work.

Wundt's position appears to me to be in the main similar to that of Munk[3] (α and β cortex function) (with the difference already alluded to in the footnote on p. 333) and of Jackson (high-level and middle-level cortex function) and to that which is presented in § 4 of this paper. Indeed my own views have been in large part formed by reflecting upon a hint originally derived from Wundt, *i.e.*, the notion of neural synthesis. But of this I have already spoken. I hasten to add that in giving this reading of Wundt's attitude *re* apperception, I do not pretend to make Wundt responsible. The misconceptions liable to arise in any transfer of abstract thought from mind to mind, have multiplied around these words—will, attention, thought, apperception—and the association of contradictory connotations round the same words in different minds, have almost destroyed their value as middle terms between mind and mind.[4] Yet I feel obliged to offer my interpretation for whatever it may be worth; it

[1] Wundt. (*Phys. Psych.*, 3rd ed. 1887. Vol. ii., p. 389, note 7). "Den seltsamen Bericht, den Alex. Bain seinen englischen Lesern von dem sonstigen Inhalt meiner psychologischen und metaphysischen Ansichten gibt, kann ich hier mit Stillschweigen übergehen. Er beweist, dass dieser ausgezeichnete Gelehrte seine Kenntniss meiner Psychologie nicht dieser selbst, sondern dem Bericht irgend eines Lesers verdankt, der die Lectüre meines Buches mit seinen Reminiscenzen an irgend einen Cambridger Theologen des 17 Jahrhunderts verwechselt zu haben scheint."

[2] Wie hat sich doch die neuere Psychologie mit der Ueberwindung auch der letzten Spuren der obsoleten Vermögenstheorie gebrüstet!" (Wundt, Phil. Stud., vi., p. 17). The term "faculty," in consequence of its adoption by the phrenological inventors, has acquired connotations that have altogether unfitted it for employment except as a polemical missile. Otherwise it often presents itself as a most suitable term to denote particular psychological properties; "memory" as a psychological faculty of a special cerebral organ is a physiological non-sense; sight, smell, as physiological faculties or susceptibilities to particular forms of excitation (*i.e.*, in a passive sense with the connotations of the French word "faculté," as implying a potentiality, *e.g.*, faculté d'agir) would be in many cases a suitable and most convenient term, if only the word had not been debauched. But as it is, the word is only fit to be used as an abusive label for psychological vagaries. Perhaps it may become disinfected by time.

[3] As regards historical order the two names should, of course, be transposed.

[4] A. Marty remarks incidentally that "es bedarf natürlich einer ganzen Reihe von Verwechselungen und Aequivocationen um von hier aus [*i.e.*, Apperception = das Bemerken] die Apperception für identisch mit dem Willen zu erklären. (Zsch. für Psychol. und Physiol. der Sinnesorgane, 1892, p. 140).

is at any rate the resultant of a strenuous "effort of apperception" excited by Wundt's own writings.

The points of *opinion* aroused in me by the careful perusal of Wundt's exposition (that in the 3rd edition of his Phys. Psychol, 1887, as well as that in the 2nd edition (French trans., 1886), are:

1st. That he altogether rejects the faculty theory of cerebral activity.

2nd. That "apperception" is no more than his label to denote high-level neural synthesis.

3rd. That Wundt's doctrine of apperception is in no opposition to the associationist doctrine of the English school (the two Mills, Bain, Spencer), but fully inclusive of that doctrine.

4th. That Wundt's frequent indentification of "Apperception" and "Will" illustrates the impossibility of dividing the supreme central process into sensory and motor. "Apperception" (the term with centripetal connotation) and "Will" (the term with centrifugal connotation), are thus distinct terms denoting one indivisible senso-motor process of "Thought."

But this is only my opinion, and I am open to correction in this somewhat obscure and dubious matter. All that I venture to assert positively is that Wundt has been mispresented to English learning by the paraphrases of Bain and of Bastian, precisely as Kant was mispresented to a former generation by the careless eloquence of Coleridge. The Kantian faculty puppet was fathered by Coleridge, not by Kant; the Wundtian faculty puppet now slipping into English literature, is equally illegitimate.

It is not possible to describe the highly abstract bearings of the Wundtian doctrine of "apperception" by any short paraphrase, or by quotations extracted from their context. It is not possible in this case to get anywhere near the author's meaning without careful study of his own writings. With the express reservation that short quotation is in this case insufficient, I will select some passages in which Wundt's meaning seems most to come to a point, although point apart from shaft is not a very convincing instrument.

The last two references taken from a more recent paper, are given to indicate what indeed may be found indicated at greater length in the Physiologische Psychologie, viz., the great extension given by Wundt to the term "Will," and his view that reflex action springs therefrom, and not *vice versa*—a view shared by many leading psychologists, among others by Delbœuf, and—taking the spontaneity doctrine in its broad sense—by Bain.

"Den Eintritt einer Vorstellung in das innere Blickfeld wollen wir die Perception, ihren Eintritt in den Blickpunkt die Apperception nennen."—Vol. II. p. 236.

"Weiterhin muss aber sogar die Apperception als der primitive Willensact angesehen werden, der bei den äusseren Willenshandlungen stets vorausgesetzt wird. Bedingung für die Ausführung einer Willensbewegung ist die Apperception der Vorstellung dieser Bewegung."—Vol. II. p. 243-4.

"Ein wesentlicher Theil der Schwierigkeiten, welche zu jener Annahme einer Entwicklung des Willens aus den Vorstellungen geführt haben, verschwindet sofort, wenn man die Apperception als die primitive Willensthätigkeit anerkennt. Von einer Zeit der Willenslatenz, in der sich erst die Vorstellungen, welche eine Beherrschung der äussern Bewegung möglich machten, im Bewusstsein ansammeln müssten, kann dann an und für sich nicht mehr die Rede sein. Die innere Willensthätigkeit ist von Anfang an mit dem Bewusstsein gegeben, da es ein Bewusstsein ohne Apperception nicht gibt, und die äussere Handlung erscheint als eine Bethätigung des Willens, welche von der inneren Handlung der Apperception nur in ihren Folgen, nicht aber in ihrer unmittelbaren psychologischen Beschaffenheit verschieden ist. Als Phänomen des Bewusstseins betrachtet besteht nämlich die äussere Willenshandlung in der Apperception einer Bewegungsvorstellung. Die wirklich erfolgende Bewegung und die daraus entspringende weitere Wirkung auf Bewusstsein und Apperception ist erst ein secundärer Erfolg, welcher nicht mehr ausschliesslich von unserm Willen abhängt: die Apperception der Bewegungsvorstellung oder der Willensentschluss kann erfolgen, ohne dass die Bewegung eintritt, sobald der Zusammenhang der physischen Werkzeuge, die bei der Bewegung zusammenwirken, in irgend einer Weise gestört ist."—Vol. II. p. 470.

"Da aber weiterhin die Contraction des Protoplasma auf den späteren Entwicklungsstufen deutlich den Character eines psychophysischen Vorganges an sich trägt, d. h. einer physiologischen

Leistung, welche zugleich von psychischen Vorgängen begleitet ist, so verlangt der Grundsatz der Continuität aller Entwicklung, dass dieser Character auch schon jenem primitiven Spaltungs—und Contractions vorgang nicht fehle: er wird in diesem Sinne als ein einfacher, von Empfindung und Gefühl, eingeleiteter und begleiteter Willensact zu deuten sein" (*Phil. Stud.*, 1889, p. 339).

"Jede Uebung besteht in der Mechanisirung ursprünglich mit Bewusstsein geübter Willenshandlungen" (*Phil. Stud.*, 1889, p. 378).

And the senso-motor view seems to me very explicitly set forth in the page on which these sentences occur:—

"In jedem Gefühl ist eine Willensrichtung, in jedem Wollen eine Gefühlswirkung enthalten . . . . das Gefühl ist das erste Stadium eines jeden Willensactes. . . ." (*Phil. Stud.*, 1890, p. 376).

I cannot pretend to have fully mastered Wundt's meaning; his treatment of apperception as the determinant of Will, and in some places his apparent use of the two terms as inclusive of each other, and above all his presentation of will as the fundamental property of living matter, are depths of doctrine to which I cannot claim to have penetrated. My reading of Wundt's adoption of "Will" as the fundamental protoplasmic neurility, is that it gives expression to the fact that the functions of living things in their lowest, as in their highest manifestions, are not *finally* explained by reference to passive properties, that "excitability" and "association"—the last words of physiology and of psychology—are not the last words of philosophy. He appears to prefer to denote his foundation concept by the active term WILL rather than by the passive term EXCITABILITY. To me the choice seems fundamentally indifferent, and that the same dead end to positive knowledge has been reached whichever term we adopt. The truth that "it is the spirit that quickeneth" seems to me as clearly apparent beyond "*excitability*" as it is in "*will*," and it is merely a matter of verbal convenience to us as students of phenomenal determinism, whether we adopt "Excitability" as our foundation concept, and say that voluntary movement, objectively studied, is reflex movement; or whether we take "Will" as our foundation concept, and say that amœboid

movement, subjectively idealised, is voluntary movement. The inspiration of all *living* matter is equally manifest to us, whether we look through man to protoplasm and see in amœboid movement the first signs of "will," or through protoplasm to man and see in conduct the highest expressions of "excitability."

ON THE DESTINATION OF THE ANTERO-LATERAL ASCENDING TRACT.

BY HOWARD H. TOOTH, M.D., F.R.C.P.

In the present volume of BRAIN (p. 215) Dr. Mott contributes a very interesting paper on the upward course of the degeneration which follows lesion of the antero-lateral region of the white matter of the cord. Some of the material on which that paper was written was brought before the Physiological Society at King's College Laboratory on March 12th, 1892. At that meeting I stated that I had experimental and anatomical evidence of similar results to those of Dr. Mott, and that I was about to bring my results before the next meeting of the Neurological Society.[1] The experiment had been made in the Laboratory of the Examination Hall in December, 1890, and it is owing to the kindness of the Laboratories' Committee that I am enabled to make the following communication.

I had drawn attention to Loewenthal's experiments and observations on dogs in my Gulstonian Lectures in 1889 (pp. 62 & 64), and I had at that time also traced the degeneration of the antero-lateral ascending tract as high in the medulla as the points of emergence of the sixth and seventh cranial nerves (*op. cit.*, fig. 14), but had failed to trace it any higher. This was no doubt owing to the initial lesion (a semi-section) being too low down in the cord, the degenerated fibres being too sparse at the higher levels to be recognised by the ordinary Weigert-Pal staining method. Dr. Mott's experiment consisted of cutting the fibres of the tract by a specially-devised knife in the upper cervical region. He thereby caused a marked secondary degeneration, which was made all the more evident by the method of staining he employed.

[1] Held at University College Physiological Laboratory on March 17, 1892.

In the experiment about to be detailed the lesion included the direct cerebellar tract as well as the antero-lateral ascending tract, and in that respect must be held to be of less value than Dr. Mott's, but owing to the height at which the lesion was made (lower medulla) the degenerations are quite unmistakeable by the Weigert-Pal method of staining.[1] The appearances resemble almost exactly those in Loewenthal's drawings.

Though I am unable to trace the fibres with certainty to their ultimate destination in the cerebellar cortex, I consider that the diagram given by Dr. Mott on p. 219 represents the anatomical relations of the antero-lateral and direct cerebellar tracts correctly, as far as their course in the medulla, pons, and central white matter of the cerebellum is concerned.

On December 6th, 1890, the following operation was performed on a bonnet monkey. The medulla was exposed by trephining the occipital bone on the right side, the circle of the trephine hole touching the edge of the foramen magnum. The remaining sharp edges of bone, and also the lamina of the atlas, were removed by the bone forceps. The dura was then carefully slit up so as to expose the medulla and inferior vermis of the cerebellum. A small dorso-ventral incision in the lateral region on the right side was then made by means of a fine cataract knife. All this of course was done under strict antiseptic precautions and under complete anæsthesia.

The animal recovered so rapidly as to be practically quite well and lively the next morning.

Sensation was tested in all forms (except the clip test) and found to be perfect.

The animal was killed thirty-four days after the operation. After hardening in Müller's fluid the medulla and cerebellum were imbedded in celloidin, and the sections were stained by the Weigert-Pal method.

Microscopical Examination.—The general appearances are represented diagrammatically in the plate, which is a

[1] Dr. Mott and I have compared our results carefully, and have agreed that the degeneration in the two series of sections are practically identical.

reduction of the diagram shown at the meeting of the Neurological Society at University College, on March 17th, 1892. The degenerated areas are indicated by black patches.

1.

FIG. 1.—Through the medulla above the level of the first cervical, and at about the middle of the lesion. The lesion is seen to be a very small one. It involves the direct cerebellar and antero-lateral ascending tracts. It has involved a very few of the fibres of the ascending root of the fifth (V.a).

2.

FIG. 2.—At the level of the lower olive. The initial lesion is no longer to be seen, the appearances being now due to true secondary degeneration. This consists of a strip extending dorsally from the funiculus cuneatus (f.c.), to ventrally the level of the nucleus lateralis (n.l.) It bounds externally the ascending fifth (V.a.), which is normal. Many normal external arcuate fibres may be seen streaming into the degenerated area to form the corpus restiforme higher up. Many degenerated fibres may be seen in the nucleus lateralis.

3.

FIG. 3.—Made through the lower rootlets of the twelfth nerve (XII.). Central canal now opened out into the fourth ventricle. The strip of degeneration is now practically divided into two parts, a dorsal portion situated externally to the funiculus cuneatus, intensely degenerated, which is the direct cerebellar tract (d.c.t.), and a ventral, situated externally to the nucleus lateralis, which is the ascending antero-lateral tract (a.t.) These two tracts are divided by a neck of healthy arciform fibres belonging to the external series.

4.

FIG. 4.—A little above the last. The degeneration of the direct cerebellar tract is becoming invaded by numbers of normal fibres derived from the external arciform group, the two together forming the corpus restiforme. The ascending antero-lateral tract consists of many degenerated fibres lying among the external arciform fibres dorsally and externally to the olive.

FIG. 5.—At the lowest part of the eighth nerve (VIII.), and the ninth nucleus and nerve (IX.)

The direct cerebellar tract degeneration is now a small patch in the dorsal third of the corpus restiforme, which is here well developed. The antero-lateral ascending degeneration is compact, and lies just dorsally to the olive.

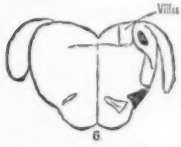


FIG. 6.—At the upper extremity of the olive. Corpus restiforme now very large, with the small direct cerebellar degeneration in its dorsal third. Antero-lateral ascending tract in same position as before. (VIII. a.) ascending root of eighth nerve.

FIG. 7.—At the level of the seventh nucleus and superior olive. The corpus restiforme is quite buried in the mass of the middle cerebellar peduncles, and still shows its small patch of direct cerebellar degeneration. Unfortunately here and in the next section there is a considerable amount of decomposition evident in the central parts of the section; this is due to the slow penetration of the hardening reagent.[1] It requires very careful comparison between the two sides to be sure which is degenerated and which is decomposition.

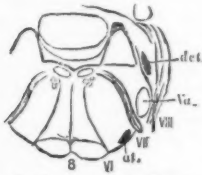


FIG. 8.—Intra-medullary course of sixth (VI.) and seventh (VII.) cranial nerves. The fibres of the corpus restiforme are at this level cut obliquely owing to their taking a direction dorsally to pass to the vermiform process of the cerebellum. The degeneration of the direct cerebellar tract can still be seen, but there is also some decomposition change in its surroundings. The brachium con-

[1] I have endeavoured to get over this by injecting the whole animal with Müller's fluid through the aorta, after washing the blood out by neutral saline solution immediate after death. The Müller's fluid, however, causes so intense a spasm of the small arterioles that the injection cannot reach the central parts. This difficulty is obviated by sending a fairly strong solution of atropine through the vessels, after washing out with saline solution, thus causing paralysis of the muscular coats of the small arterioles.

junctivum, or superior peduncle of the cerebellum, is now seen in somewhat oblique section with the fibres of the corpus restiforme curling round it. The two brachia conjunctiva form the side walls of the upper part of the fourth ventricle, stretching across the ventricle and forming its roof is the velum medullare anterius, dorsally to which is the lingula or upper extremity of the vermiform process. The velum medullare anterius consists of white fibres apparently arising from the brachia. In the angle formed by the brachium with the lingula is a small mass of degenerated fibres cut in transverse section. These are the bent-back fibres of the antero-lateral tract. So in this and the succeeding figures, except the last, we see the two limbs of the loop of the antero-lateral ascending tract—one ventral and the other dorsal.

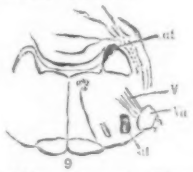


FIG. 9.—At the level of the upper part of the loop of the seventh nerve. The brachium conjunctivum is now well differentiated; it consists of a main body of transversely cut fibres, somewhat pyramidal in section, with a sort of cap of obliquely-cut fibres applied to its apex and mesial aspect. The set of fibres on the apex is deeply degenerated (a.t) as stated in the last paragraph, the mesial set is continuous with the velum medullare anterius. Hooking round the brachium are the fibres of the restiforme body, which appear to go to the lingula, and many are degenerated, the direct cerebellar tract (shown by dotted lines). The ventral part of the antero-lateral tract (a.t) is beginning here to take an oblique course ventro-dorsally towards the trunk of the fifth nerve, which is here just sending off the fibres which are to make up the so-called ascending root.

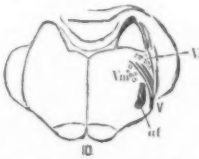


FIG. 10.—At the level of the fifth nerve (V.) The disposition of the dorsal degeneration of the antero-lateral tract remains the same, being at the apex of the pyramidal section of the brachium conjunctivum. The fibres of ventrally-disposed part (a.t) are now cut about longitudinally, and are

seen to be taking a direction as if to intersect the course of the fifth nerve. Vs. sensory and Vm. motor nuclei of fifth.

FIG. 11.—Just above the fifth. Dorsal part of antero-lateral tract, now disposed more on the outer aspect of the brachium than before, the ventral part having now hooked round the fifth is approaching the dorsal part, the intervening fibres being those of the lateral lemniscus (ll). Lemniscus or fillet (l). Brachium conjunctivum (bc.)

FIG. 12.—Section through upper pons. The two degenerations are now quite close together. Vd. descending root of fifth. Pl. posterior longitudinal fibres.

FIG. 13.—At the level part of the lowest point of exit of the fourth nerve (IV.) This shows the extremity of the loop formed by the antero-lateral ascending tract. The degeneration consists now of one strip applied externally to the fibres of the lateral lemniscus which lies between it and the brachium conjunctivum.

THE FUNCTION OF THE SYMPATHETIC GANGLIA.

A Reply to a Criticism by Dr. Friedric Vas.

BY W. HALE WHITE, M.D.,
Physician to Guy's Hospital.

I HAVE in various papers[1] urged that some of the sympathetic ganglia in adult man are functionless organs and are (like the appendix cœci or the coccyx) the remnants of structures having important functions in the lower mammals. With regard to the superior cervical ganglion this result was arrived at for the following reasons:—(1) The human adult superior cervical ganglia vary as much in size as do those of the largest and smallest of other mammals, but the size of the ganglion in other mammals than man varies directly as the size of the animal. It is well known that degenerate organs are particularly liable to vary considerably. (2) The superior cervical ganglia taken from forty-nine adult human beings were examined. Of these only one showed entirely normal nerve cells. In the others the cells were more or less shrunken, non-nucleated, granular and irregular in outline. They were also very strongly pigmented. The result of these changes was that often the cells were reduced to mere minute masses, usually of a bright yellow colour, lying quite free in the centre of the cell spaces, without processes, and as far as could be seen, without any connection with nerve fibres. But the superior cervical ganglia taken from five children showed that the cells were large, they filled their spaces, the nucleus and nucleolus were distinct, the protoplasm was non-granular, and there was no trace of pigmentation. The cells of the superior cervical ganglia taken from ten human fœtuses were like

[1] *Journal of Physiology*, Vols. 8 and 10; *Guy's Hospital Reports*, Vol. 46; *Medico.-Chirurg. Soc. Trans.* Vol. 68; BRAIN, Pt. 51.

those from the children. The facts appear to warrant the belief that the superior cervical ganglion although relatively prominent in the fœtus atrophies with the growth of the individual. (3) I examined the superior cervical ganglia in sixty-eight mammals lower than man. Fifteen of these were monkeys and in four of them the cells were more or less like those just described as found in human adults, but in the remaining eleven monkeys and in the fifty-seven other mammals the nerve cells were well formed with a good nucleus and without any granular change, they filled their capsules and generally showed absolutely no pigmentation, although occasionally a little was present. Several of these mammals had been kept in the zoological gardens for many years and it was well known that they were very old, but yet their cells were quite different from the minute masses of granular pigment without nuclei which represented the nerve cells in the ganglia taken from old men. These results point also to the fact that in the mammals lower than man the ganglia are functionally active. (4) The examination of thirty human adult semilunar ganglia, of three from children, and of eighteen taken from mammals lower than man, for the same reasons as already urged for the superior cervical ganglion, appeared to show that in adult man the cells of the semilunar ganglia were inactive, but that they were functionally active in mammals lower than man. (5) The thoracic ganglia of twenty-four human adults were examined and it was found that in many of them the cells were large, distinct, neither granular nor atrophic, but in some these changes were slightly marked. It was concluded that in adult man the thoracic ganglia were only slightly degenerate. (6) Thirty-six gasserian ganglia taken from human adults were examined; in all the nerve cells were well formed with a distinct nucleus and nucleolus, and they filled their capsules. A few of the cells showed some granular pigment, but the cells themselves were not in any way atrophied. (7) Six lenticular ganglia from the orbit were examined. The cells all filled their capsules, were distinctly nucleated, but were neither granular nor pigmented. (8) Twelve spinal ganglia were examined. The cells never showed any of the extremely degenerate

appearance so common in the superior cervical ganglia, but often they were pigmented. (9) The cardiac ganglia were examined in ten patients; the nerve cells showed no shrinking, granular change, nor pigmentation. (10) It was shown that when the superior cervical and semilunar ganglia are invaded by disease no symptoms are produced except those which can be explained by the pressure on the nerve fibres passing through the ganglion, and that in the case of all diseases which have been ascribed to intrinsic lesions of the ganglia such a pathological explanation will not bear critical examination.

Thus it appears that the gasserian ganglion, the lenticular ganglion, and the cardiac ganglia are, in adult man, probably functionally active, and that most of the cells of the posterior spinal ganglia are also active. It will be noticed that the large number of 270 ganglia in all were investigated, and of each of these several sections were examined.

Dr. Friedrich Vas[1] has recently criticised the views I have put forward, and I propose to answer his objections.

Meyer, writing in 1876, suggested the view that some of the sympathetic ganglia in adult animals merely represented remains of structures which were of embryonal importance, giving as a reason that they could be excised from dogs without particularly affecting the animal from which they were taken. Vas urges rightly that we must not conclude therefore that they are functionless, for, as he says, the spleen can be excised and the animal will after a time be no worse for it. He might have also added that part of the brain and one kidney may be taken out without producing any permanent effect, but in all these cases it is quite certain that the excised organs have some function. But this just criticism by Vas of Meyer does not affect my contention at all, for an essential part of my view is that in dogs the sympathetic ganglia have some distinct function, but that in adult man this has disappeared. Meyer's argument is quite worthless either way, for while it certainly does not prove that the ganglia have no function, it certainly does not prove that they have any.

[1] *Allgemeine Wiener Medizinische Zeitung*, N. 45, 46 und 847, 191.

Vas states that I attribute great importance to pigmentation of the nerve cells of the ganglia, to their not filling their capsules, to their protoplasm being granular, and to the absence of the nucleus and nucleolus, as showing that they are a degenerate type of cell. This is not quite correct. I do not attach great importance to pigmentation as evidence of degeneration, I express myself very doubtfully on the subject. Thus (*Journal of Physiology*, vol. x., No. 5, p. 343) I say, "This may perhaps mean that we ought to regard pigmentation as evidence of degeneration." I never conclude because the cell is pigmented that therefore it is degenerate. It so happens that when from other reasons the cell is probably to be regarded as degenerate it is usually pigmented, and therefore when describing these cells I describe them as "granular pigmented atrophic" cells. I do not know the meaning of the pigmentation.

Vas goes on to say that I allow that sometimes nerve cells which show all these changes are nevertheless functionally active. I do not state this, and I never intended to convey such a meaning. Probably he refers to the fact that when the cells, which in any ganglion show these changes, are very few, I draw the conclusion that almost all the ganglion is functionally active, the only functionless parts of it being the few degenerate cells. Its activity is is in fact just beginning to die out. This is the conclusion at which I arrived with regard to the thoracic ganglia and those on the posterior roots of the spinal nerves. In particular Vas mentions that I say that sections of the gasserian ganglion may contain one or two granular pigmented cells, and yet he objects that I do not conclude from this that the gasserian ganglion is in adult man an embryonal remain. Of course I do not. The very fact that the vast majority of its nerve cells were like ordinary healthy nerve cells goes to prove that nearly all the nerve cells are functionally active and I distinctly state those few which were altered were never atrophic. From these facts I think I am justified in my conclusion that the ganglion as a whole is functionally active.

He next states that there are two questions to be asked

in connection with my view. In the first place, if the sympathetic ganglia are embryonal remains (embryonale Reste) how comes it about that they continue to increase in volume after birth. My answer to this is that I never said that the ganglia were embryonal remains. I said that those ganglia which in adult man show degenerate cells (*e.g.*, the superior cervical) were "atrophied degenerate organs like the coccyx or appendix cœci." Embryonal remains are structures which in the life of the fœtus were of great vital importance to it, such as the umbilical cord or the hypo-gastric arteries, and they will, directly after birth as their function then ceases, begin to gradually atrophy. But organs which are vestigial structures, representing in man organs which in the lower animals were important, and which are gradually dying out, will not necessarily begin to atrophy, because life changes from intra-uterine to extra-uterine. Such organs may continue to grow (although not to anything like the extent to which they do in the lower animals) for a long time in the life of the human being. For example, the coccyx, an undoubted degenerate organ, does not begin to ossify till birth, and the last centre of ossification does not appear till about the fifteenth year. Therefore, it does not by any means follow that because the superior cervical ganglion grows a little after birth that my view is incorrect.

The second question asked of me is, how is it possible that, if in the human adult the superior cervical ganglia have lost their function, those of the thorax in organic connection with them have maintained theirs? Surely there is no reason why they should not. We do not argue that the coccyx of man cannot be a degenerate organ because the lumbar vertebræ are well developed. For all we know, in creatures in whom the superior cervical ganglia are well developed, they subserve functions quite different from those of the thoracic ganglia—in fact, this is very likely, for the superior cervical ganglion is a collateral ganglion and the thoracic are lateral ganglia.

Vas next gives as a reason why the superior cervical ganglia cannot be looked upon as vestigial degenerate organs

that they show no fatty degeneration. But this proves nothing. Numbers of degenerate organs, which in man undoubtedly represent structures which in the lower animals were of great importance, show no fatty degeneration; for example, the appendix cœci and the coccyx.

He goes on to say that the fact that the nerve cells of what I regard as the degenerate ganglia are very much smaller than their capsules is to be explained by artificial shrinking, due to the method of preparation. I am sure this is not so, for three reasons: firstly, the lenticular and gasserian ganglia in which it was not present were prepared in precisely the same way as those ganglia, as the superior cervical, in which most of the cells showed it; secondly, many ganglia, such as the thoracic and posterior spinal ganglia, showed this shrinking in some cells but not in others; thirdly, the cells can often be seen shrunken in superior cervical ganglia, when they are examined fresh directly they are taken out of the body. Also after the examination of hundreds of sections I feel quite sure he is wrong when he says that the nucleus and nucleolus of the human superior cervical ganglion are intact and easy to make out. I have examined hundreds of sections from man in which this is not the case.

Vas next urges that my view cannot be correct because if it were the ganglia of the rabbit should be different from those of man, which he says is not the case. In the first place it should be noticed that he does not say what ganglia he examined in connection with this point, but judging from his paper he does not appear to have investigated any but the superior cervical, so I conclude that he refers to these. In the next place it would not follow that because the superior cervical ganglion of the rabbit resembled that of man that the ganglion did not represent an organ that in man had lost a function which was important in the lower animals, for it is quite possible that in both man and the rabbit the ganglion might be a degenerate vestigial remain. For example, both in man and in a Manx cat the tail is a rudimentary, but we do not therefore conclude that the rudimentary tail of man is not the degenerate representative of the tail of the lower animals. It so happens

that although my published papers contain an account of the examination of the superior cervical ganglion of sixty-eight mammals lower than man, not one of these animals is a rabbit; so since reading Vas's paper I have examined a number of sections of both superior cervical ganglia of a rabbit, and I can confidently say that very nearly all the cells are well formed nerve cells, showing none of the atrophic condition so common in the cells of the same ganglion taken from adult man. Some of the other mammals show rather larger superior cervical ganglion cells than the rabbit, but almost all those in this animal are entirely different from those quite commonly found in adult man. If I am correct and Vas is wrong we see that the rabbit forms no exception to the rule that the cells in this ganglion of animals lower than man appear to show that it is a functionally active organ, although in man its cells are inactive and degenerate.

Vas further says that on my view the cells of the sympathetic ganglia of the frog ought to be functionless, for they are strongly pigmented, but I have already pointed out that I do not regard mere pigmentation as evidence of degeneration.

He next goes on to argue that if the nerve cells of the superior cervical ganglion in man are atrophic degenerate structures, you ought, by cutting the nerves either side of the ganglion, to be able, in rabbits, to cause the ganglion cells to have a similar appearance to that which I describe as the atrophic condition in man. Accordingly he cut the nerve above and below the ganglion in a rabbit and killed the animal fourteen days after. But I maintain that it is obviously unfair to contrast the condition artificially produced in a rabbit in fourteen days with that which has probably been produced in man by forces operating for countless ages. What would be thought of the man who, because he could not in fourteen days, after cutting all the nerves going to the tail of a long-tailed monkey, make it shrink to the size of the coccyx of an adult man, concluded that the coccyx in man did not represent the tail of the monkey. We need not therefore, I think, stop to discuss Vas's description of the results he obtained except to remark

that it is strange that he found that only the cells in the periphery of the ganglion were degenerated after section of the nerves at the side of it.

Vas admits that in some cases he found the human superior cervical ganglia to be such as I have described, but he says that all the patients presenting this condition had arterio-sclerosis of their vessels, and he is inclined to draw the conclusion that there is a casual connection between the degeneration of the ganglion cells and the arterio-sclerosis. There can, I think, be no doubt that this view is wrong. A reference to the lists published in the *Journal of Physiology* (vol. viii., No. 2, and vol. x., No. 5) will show that the forty-nine adults whose superior cervical ganglia I examined died of the most various diseases, and that no relationship can be traced between the disease from which the patient suffered and the condition of the cells of the superior cervical ganglion. In connection with this subject I may mention that Da Costa and Longstreath (*American Journal of Medical Sciences*, July, 1890) record eight cases of chronic Bright's disease, associated with what they consider to be pathological changes in the semilunar ganglia. The ganglion cells were pigmented and granular, neither nucleus nor nucleolus could be seen, and they were often shrunken. These changes have no value in Bright's disease, for, as I have elsewhere recorded (*Guy's Hospital Reports*, vol. xlvi., and *Medico-Chirurgical Society's Transactions*, vol. lxviii.), I have often found these changes in these ganglia even when no chronic Bright's disease has been present.

Lastly, I would point out that Vas's researches cover too little ground. He appears to have examined no other ganglion than the superior cervical, and that only in rabbits and in man. He does not say how many specimens he looked at. No children, nor fœtuses were investigated, and even in arterio-sclerosis, about which he says so much, the superior cervical ganglia were the only ones examined, and even they were looked at in three cases only.

It will be seen, therefore, that in spite of Vas's criticism I maintain my view, which is given at the beginning of this paper, as to the meaning of the sympathetic ganglia in adult man.

HYALINE DEGENERATION OF THE SPINAL CORD.

BY WILLIAM BULLOCH, M.B., C.M.ABERDEEN,

From the Pathological Laboratory, University of Aberdeen.

FROM the scarcity of literature on this subject it would appear that hyaline degeneration in the spinal cord is not a common condition. Most of the recorded cases have occurred in conjunction with syringomyelia. Anna Baümler (*Deutsch. Archiv. für Klin. Med.*, 1887) refers to several cases; among them one by Hutin, in which the dorsal cord is described as being of a glassy appearance.

Langhans (*Virchow's Archiv.*, Bd. lxxxv.) quotes the case of a girl, aged 19, who had a syringomyelic cavity in her cord. The cavity had been formed by the burrowing of a homogeneous gelatinous mass between the elements of the spinal cord, so as to compress the latter and lead to their degeneration.

A full account of hyaline degeneration, accompanied by syringomyelia, is given by Berkeley (BRAIN, 1890, vol. xii., p. 460). In his case, besides the fissure the cord was found extensively infiltrated by some hyaline material, which was in greatest amount in the neighbourhood of the vessels. Several vessels were also found filled with material closely resembling the hyaline masses. In some parts where the hyaline had exuded in large amount, the axis cylinders had been compressed and had undergone degeneration. After a careful research into the reactions and site of the hyaline material, Berkeley comes to the conclusion that the "new product is a peculiar exudation from the blood vessels, which, after absorbing and destroying to a certain extent the surrounding tissues, underwent itself a further change and broke down with cavity formation, or in some places with rupture of the vessels and consequent hæmorrhage."

In the case about to be related the changes found in the cord strikingly resembled those found by Berkeley in his case, except that in the present the changes were much more intense. The case at first appeared to be a typical one of acute ascending paralysis, but there appeared later other symptoms which compelled a reconsideration of the diagnosis, and then it became apparent that the case was very complicated from a diagnostic point of view, and atypical.

A case of acute ascending paralysis has been recorded by Eisenlohr (*Virchow's Archiv.*, 1878, Bd. lxxiii. p. 73), in which, at the level of the third cervical nerve, a mass was found which was strongly stained with carmine, and it showed fine slits in it but no trace of cellular or other elements. The same material was also found in the pia mater, especially around vessels which ran into the substance of the cord, and also in the commissural region near the great vessels there. A similar appearance was also found in the upper lumbar region, and also in the medulla and pons, although to a less extent. Duchenne ("Selections from the Works of Duchenne," edited by G. V. Poore, M.D., New Sydenham Society, 1883, p. 129) relates a case of "*Diffuse general sub-acute spinal paralysis*," in which a *post-mortem* examination was conducted by MM. Charcot and Joffroy. They found the "cervical enlargement of the cord enveloped in the posterior two-thirds in a kind of fibrous muff the texture of which bore no small resemblance in aspect and consistence to the tissue of the cornea. . . . In the back horns the nuclei were embedded in an amorphous finely granular semi-transparent soft material. In other parts of the back horns the nuclei had disappeared and the amorphous substance there constituted centres more or less voluminous, clearly defined and circumscribed by a kind of non-resisting membranous zone."

Charcot ("Lectures on the Diseases of the Nervous System," second series, p. 166, New Sydenham Society, 1881), refers to a case of cervical spinal myelitis with pachymeningitis, in which "three long and narrow canals were found in the cervical enlargement parallel to the long axis of the cord. Two of the canals were found mostly filled

by an amorphous, transparent, finely granulated substance, which at certain points had become disintegrated and had left in its place more or less extensive lacunæ with more or less irregular borders. This same finely granular substance slightly condensed formed the parietes of the foci, and without any well-marked line of demarcation became continuous with the adjacent tissue, which itself for some distance presented the characters of granular degeneration."

The appearances described by Charcot correspond to a certain extent to those which were found in the case to be described, for in many places the hyaline substance with high magnifying powers appeared to be finely granular, and it is probable that the transparent amorphous material described in the recorded cases was of the same nature. In the majority of the cases thickening of the vessel walls and the transformation of the latter into some hyaline material was observed.

The case was remarkable, inasmuch as the nervous symptoms came on after the patient had been suffering from profound anæmia of the pernicious type. Spinal degenerations in the course of pernicious anæmia, especially in the posterior columns, appear to be not infrequent, but the symptoms are masked by the profound general weakness.

Lichtheim (*Berlin Klin. Wochenschrift*, 1889, Oct. 14) found abnormalities in the cord in every case he examined. In half of the cases apoplectic extravasations had occurred, giving rise to minute sclerotic foci, while in the other cases the posterior columns were found widely degenerated.

The clinical history of the case is briefly as follows:—

W. B., aged 51, a farm labourer, was admitted into the Royal Infirmary, Aberdeen, on July 10th, 1890, under the care of the late Professor Smith-Shand. He complained of shortness of breath, and swelling of the feet and face. He said he had enjoyed good health until two months previous to admission, and he was of steady habits, there being no history of alcoholism or syphilis. His trouble had come on gradually. On examination he was found to be fairly well nourished, but showed considerable œdema of both feet and ankles and also of the face. He was profoundly anæmic,

the face presenting a pale lemon yellow colour. The temperature was normal and also the urine, except that the colour of the latter was very pale.

There was a loud blowing systolic murmur, heard loudest at the apex and transmitted round to the left axilla, where it became inaudible. The second sound at the pulmonic cartilage was accentuated. There was no evidence of hypertrophy of the heart, and the pulse was weak.

Respiratory and alimentary systems appeared to be normal. Liver dulness normal. Splenic dulness was found to be increased.

Examination of the blood showed that there was great diminution in the number of the red blood corpuscles, which at the same time were much altered in size and shape. Microcytes and poikilocytes were found in abundance, and a few of the corpuscles known as Eichorst's corpuscles were also seen.

Flammiform hæmorrhages were present in both retinæ, but no optic neuritis.

The nervous system appeared to be healthy except that in his intellectual functions the patient was slow and heavy of comprehension, and he had occasional delusions. The motor functions were undisturbed, although he was rather awkward and unsteady when he got up. At the time this was attributed to his great weakness, but in the light of the further development of his trouble it may have been the commencement of the spinal disease which manifested itself later on. From the state of the blood and the profound asthenia, coupled with the colour of his skin and the hæmorrhages in the retinæ, the diagnosis of anæmia of the pernicious type was made, and he was put upon five-minim doses of liquor arsenicalis three times daily, and he was carefully dieted. Under this treatment he improved greatly, the blood becoming richer in corpuscles and the form of the latter being normal. The awkwardness of gait also disappeared, and with the return of strength the swelling of the feet also.

On the 12th of September, after a two months' stay in the Infirmary, he was sent to the convalescent hospital for a short stay previous to his return home. During his stay

here he became the subject of frequent hallucinations, believing that people were speaking to him and annoying him during his sleep. Notwithstanding these peculiarities he was perfectly manageable and caused no disturbance, and he regained his strength so much that he walked several times to the hospital to report himself.

In the third week of his stay at the convalescent hospital the superintendent observed that he exhibited great inco-ordination of gait, and that if he stood for a short time his legs gave way beneath him. The gait was when seen at this time ataxic, and he was unable to walk in a straight line. Romberg's symptom was also present. In addition to the ataxia, however, there was distinct paresis of the legs, and this became so great that on the 2nd of October, 1890, he was again removed to the hospital. On the following day the paresis had involved the whole of the lower extremities, and by next day the paresis had become paralysis. The limbs were flaccid, and superficial and deep reflexes were abolished. Bladder and rectum were unaffected. About the 5th of October the hands and arms became affected, and great inco-ordination for all movements was observed, rendering the patient absolutely unfit to feed himself or do almost anything with his hands. On the 7th paresis was noticed, and by the 9th of October both arms were completely paralysed. His mental condition was one of apathy and listlessness, and his memory began to fail so that he called people by their wrong names although he could recognise them perfectly.

His condition, which was one of complete motor paralysis of upper and lower limbs, remained practically unchanged for some weeks. The tactile sensibility was diminished. The sense of locality, position of the limbs, the muscular sense and the thermal sense were normal.

He sometimes passed fæces and urine involuntarily, but this appeared to be due to his general failure in intellect rather than paralysis.

His temperature, which had remained normal, used sometimes to rise as high as 104° F., and then come slowly down during the next two or three days.

His appetite became capricious, and he suffered greatly

from thirst. He had several severe attacks of diarrhœa, which lasted for three or four days at a time. On two or three occasions his urine required to be drawn off by the catheter. No pain was complained of during the entire course of the illness, the patient always describing himself as feeling better.

Towards the end sensation appeared to be affected, but the patient was so stupid that rational answers could not be elicited from him. His memory, which had been defective, was entirely lost, and his sleep was much disturbed so that he used to cry out and threaten vengeance on the neighbouring patients. Bed sores formed on his hips and sacrum, and they were with difficulty dressed and kept clean.

About the beginning of December, 1890, it was evident that he was much weaker and indeed worse in every way. He seemed to have considerable difficulty in swallowing, and his respirations at times were loud and stertorous. On the evening of the 14th of December he became comatose and died on the 15th.

Necropsy, December 17th, 1890.

Height 65½ inches; circumference at shoulders 44 inches; *post mortem* lividity slight on dependent parts. A patch of reddish discolouration on right side of thorax due to an old burn.

Rigor mortis absent. General nourishment poor; pupils dilated and equal in size.

Large bed sore over the sacrum; on the left buttock was a slough 1¼ inches in diameter, and a sore over the right trochanter, 1 inch in diameter, partly cicatrised. Face and thighs jaundiced.

Heart weighed ten ounces; *pericardial sac* contained one ounce of straw-coloured serum. Aortic valves .8 inches in diameter, mitral 1.2 inches, pulmonary 1.1 inches, tricuspid 1.3 inches.

Left lung weighed twelve ounces. Thirty ounces of straw-coloured fluid were found in left pleural cavity, and on the outer surface of both lobes was a layer of recent fibrinous lymph. Lower lobe in a state of congestion.

DESCRIPTION OF FIGURES, TO ILLUSTRATE PAPER ON "HYALINE DEGENERATION OF THE SPINAL CORD."

By William Bulloch, M.B., C.M., Aberdeen.

Fig. 1.—Section of the cord at the level of 4th cervical nerve, showing large cavity opening to the front by a wide opening and extending posteriorly and also into the right lateral half of the cord. The walls of the cavity are clear and hyaline, vessels dilated and extravasations to be seen in the neighbourhood; vessel walls hyaline and thickened. Grey matter is not distinguishable.

Fig. 2.—Section at the level of 5th cervical nerve. Cavity not so large and degeneration not so extensive as in Fig. 1. Vessels are all dilated and walls much thickened.

Fig. 3.—Section at the level of 7th cervical nerve. Hyaline degeneration chiefly in posterior columns, but also in lateral columns. All the vessels have thickened hyaline walls. No ganglion cells can be seen in grey matter. Central canal region occupied by a mass of hyaline. At the bottom of anterior fissure the artery of the fissure seen surrounded by hyaline material.

Fig. 4.—Section at the level of 5th dorsal nerve. Degeneration chiefly limited to posterior columns. Few cells to be seen in the grey substance. Hyaline mass in central region.

Fig. 5.—Section at the level of 12th dorsal nerve, showing degenerated tract limited chiefly to the postero-internal columns. Large mass of hyaline in region of central canal, with ill-defined cellular elements in its centre.

Fig. 6.—Section in the upper lumbar region. Central hyaline mass well defined. Degenerated tract in posterior column small.

[*Autotyped from original pencil drawings, by Thévoz & Co., Geneva.*]

Fig. 1.

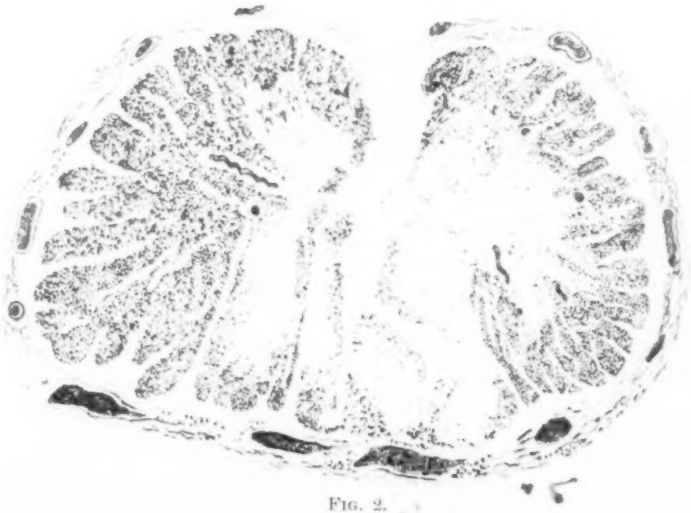


Fig. 2.

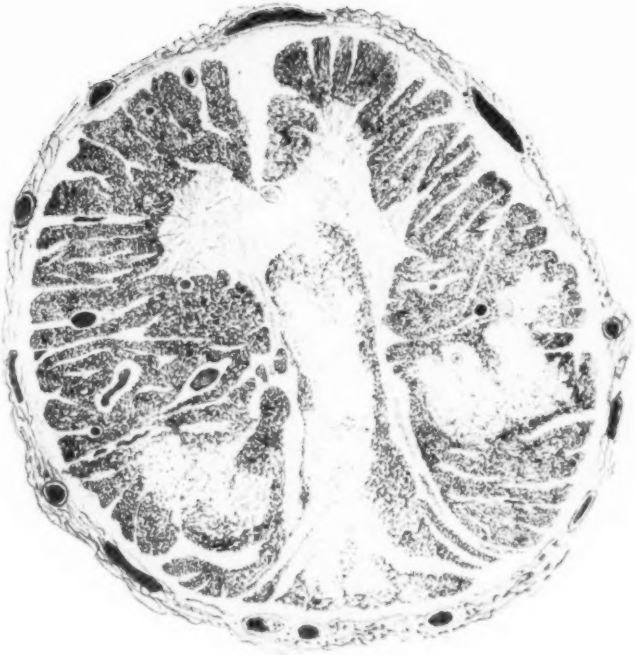


Fig. 3.

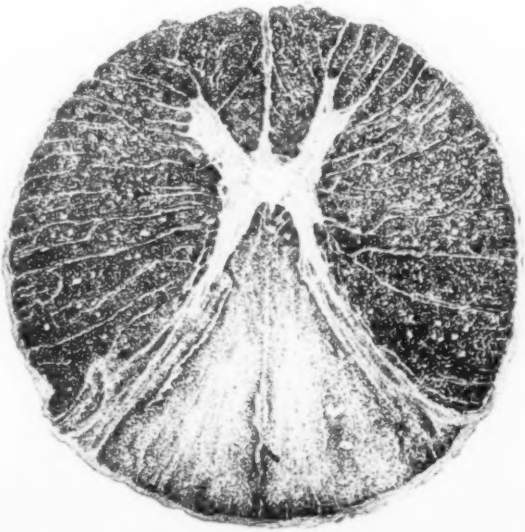


Fig. 4.

Fig. 5.

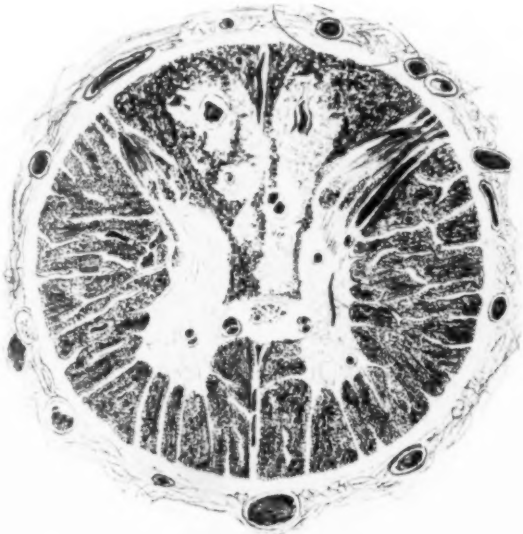


Fig. 6.

Right lung weighed 1lb. 5oz., and the pleural cavity contained ten ounces of serum. Old fibrous adhesions of pleura to diaphragm and costal parietes on the outer side. Lower lobe congested and œdematous.

Liver weighed 3lb. 11oz., and was congested and nutmeg-like in appearance. Gall bladder contained two ounces of bile.

Kidneys appeared to be normal, and were five ounces in weight respectively.

Spleen weighed nine ounces; consistence appeared to be softer than normal.

Nervous System.—One ounce of straw-coloured serum in subdural space of the brain. Dura mater adherent to vetex of brain, especially along the margin of longitudinal fissure. Pia cloudy over vertex. The brain presented nothing abnormal on the surface. The vertebral, basilar and middle cerebral arteries were slighly atheromatous.

Spinal cord.—Dura appeared healthy, but the cord itself was distinctly softer than normal, especially at a point extending from the third to the fifth cervical nerves. Here the contour of the cord was much altered, and the substance appeared to be diffluent, so that when cut into at this level a cavity was seen which at its lower part was more of the nature of a fissure extending deeply into the substance of the cord so as almost to divide it in two.

The *cauda equina* appeared healthy.

The entire brain, pons, medulla, cord and parts of the peripheral nerves were hardened in Müller's fluid and afterwards in Ehrlitzki's fluid preparatory to the Weigert hæmatoxylin process. After hardening, some of the pieces were dehydrated and embedded in celloidin. All the sections were cut in the ordinary freezing microtome. As regards the staining reagents, many were tried. Weigert's method and Pal's gave good results as far as showing the degenerated tracts, but the finer changes were seen best in specimens mounted in Färrant's medium or glycerine.

Osmic acid $\frac{1}{4}$ per cent. yielded splendid results, especially after colouring a little with $\frac{1}{4}$ per cent. watery solution of eosin or carmine.

Picrocarmine and logwood with eosin clearly demonstrated the nature of the changes, as did also many of the anilin colours, especially anilin blue, either alone or after treatment with eosin. Sahli's methylene blue method also gave good results.

Microscopical examination of nervous system.—In the cervical region, sections at the level of the broken down cavity showed that profound changes had taken place. The cavity opened by a wide aperture with rugged uneven edges towards the front. Below the cavity was more defined and presented more the appearance of a fissure. The anterior horns were fixed widely apart, and the cleft extended through the commissure into the posterior columns, which it traversed almost completely to the periphery. At one part a lateral branch from the cavity passed into the right lateral region of the cord through the grey matter in this situation.

Membranes.—The pia mater was enormously thickened and most of the blood vessels distended to their utmost with blood corpuscles, so as to give almost an appearance of cavernous sinuses. Many of the blood corpuscles could be seen lying in the walls of the vessels, and also in the neighbouring tissue beyond. Besides the distension of the vessels the walls were very much thickened, and of a homogeneous glassy-like appearance. The thickening was most apparent in the middle and outer coats, but the inner coat also participated in the change. At certain points the walls of the vessels had given way and allowed an extravasation of blood corpuscles into the neighbouring tissues. In most of the vessels masses of some clear, finely granular material could be seen, and the same appearance was frequent in the neighbourhood.

Round about the cord there could be seen a clear hyaline layer of the same material. This was evidently the layer of pia lying next the cord, which had undergone some transformation. On staining with eosine or carmine this layer was seen to be of a deep pink colour, while the rest of the pia was of a much lighter tint. The hyaline-like masses in the vessels were also stained very deeply.

This layer of deeply-stained tissue was continuous with

the septa, which were prolonged into the cord, and which also were deeply stained by carmine. The septa were much thicker than normal, and were composed of some transparent, finely granular material. In most of them enormously thickened and distended bloodvessels could be seen, and in the neighbourhood of these the hyaline material was present in large amount.

Cervical region.—On examining the substance of the cord it could be seen that the new hyaline element had been infiltrated between the elements of the cord and compressed the axis cylinders, leading to their degeneration. Between and around the axis cylinders all over the cord the hyaline could be seen in great abundance. In the posterior columns the hyaline had accumulated to an enormous extent, and large masses of it could be seen with broken-down axis cylinders in the neighbourhood. Here and there in the lateral columns, but having no systemic distribution, the hyaline material was in large patches, the axis cylinders having completely disappeared. In the grey matter, which was almost unrecognisable, the hyaline was abundant. No trace of any nerve cells could be seen.

The changes in the nerve fibres were seen in different stages. Where the hyaline was in small amount the fibres appeared irregular in outline, and there was no distinction between the axis cylinder and the myelin sheath. These stained deeply with osmic acid. Others were seen only lightly stained, and they were smaller in size and full of black granules, whilst the furthest stage appeared to be the conversion of the fibre into a shrivelled granular mass, which was surrounded completely with the hyaline material. Here and there in the degenerated tracts blood corpuscles could be seen which had exuded from the distended vessels.

At the level of the fifth cervical nerve the cavity was much smaller, and, in fact, was a split in the cord. The edges of the split were lined with hyaline. Descending the cord it was found that the changes were not so marked.

A section at the level of the seventh cervical nerve showed that the well-defined patches of degeneration which existed higher up in the lateral columns had disappeared. A few

healthy axis cylinders could likewise be seen in the posterior columns, although at certain places there were still large masses of perfectly hyaline stuff to be seen throughout the section. The commissural region was completely filled with the hyaline, which was continuous with a large hyaline mass lying at the bottom of the anterior median fissure around the artery of the fissure.

In the midst of the commissural hyaline mass the central arteries could be seen with thickened transparent walls, and full of blood corpuscles. Indistinct cellular elements could also be recognised here and there in the mass.

Dorsal region.—Here the changes were still less marked. The grey matter appeared more normal than in the upper regions of the cord, but still no healthy ganglion cells could be seen. They were converted into granular masses in which no nucleus could be seen. The changes in the commissure were similar to the above except that they were much better defined, so that the mass in this region looks like a minute tumour to the unaided eye. In the upper dorsal region the posterior columns were still widely degenerated except along the margin of the posterior nerve roots, where many healthy axis cylinders could be seen.

In the lower dorsal region the degeneration was still less marked. On each side of the posterior median septum next the periphery of the cord there was a triangular area, which was not infiltrated with hyaline.

Still further down the triangular area increased in size, so that the degenerated tract occupied a site corresponding to the distribution of the internal posterior and the postero-median arteries, but extending up to the commissure. The central mass is well defined, and in it, besides the hyaline, there can be seen numerous cellular elements.

In the upper *lumbar region* the degeneration in the posterior columns was limited to a small area on each side of the posterior median septum. In the middle of the hyaline degenerated area in several of the sections the distended and thickened postero-median artery could be seen. A few ganglion cells could be seen in the anterior cornua, but they were limited to the antero-lateral, postero-

lateral and central groups of cells, and in each of these groups the number of cells was much diminished compared with normal. The cells were likewise merely granular masses, which insensibly merged into the hyaline matter surrounding them.

The pia was still much thickened, and most of the septa enlarged and homogeneous. The mass in the central region was still well defined like a small tumour. The nerves of the cauda equina were also involved in the change, although no degeneration of the fibres had taken place. The vessels were all much thickened and distended with blood corpuscles, the septa between the fibres were also enlarged and homogeneous, and small quantities of the hyaline could be recognised throughout the bundles. At the tip of the spinal cord the canal was much widened and filled up with cellular elements, and also with the hyaline. The degeneration in the posterior columns became unrecognisable about the fourth lumbar nerve.

Sections of the peripheral nerves appeared to be normal. Medulla and pons also appeared healthy, and microscopic examination of the brain revealed nothing. Above the cavity in the cervical region the cord was not much altered, except that pia and some of the septal vessels were thickened. There was no ascending degeneration to be seen.

Summary.—The exact nature of the changes was by no means clear. The hyaline material had probably exuded from the blood vessels and had found its way among the nervous structures of the cord, surrounding them and leading to their destruction. The ganglion cells were converted into granular masses in some instances, while in others they were represented by hyaline masses the shape of the original ganglion cells. That the hyaline material was an exudation from the vessels seems apparent from the fact that it was in greatest amount in the neighbourhood of blood vessels, that the vessel walls were hyaline, and that hyaline masses were found inside the vessels.

In regard to the material itself nothing very definite was made out.

Histologically it was deeply stained by carmine, logwood,

and especially the anilin dyes. A differential stain, which showed the ramifications of the hyaline among the nervous structures to perfection, was obtained by staining the sections in a watery solution of anilin blue, and then washing them in a weak watery solution of eosin. The hyaline material all through the section was stained bright blue, the remaining structures being pink for the most part. The walls of the thickened vessels were blue, while the blood corpuscles in their interior were pink.

Anilin blue alone stained the hyaline of a deep blue colour, while the healthy parts were of a much lighter shade.

Gentian violet gave no differential colour reaction such as is obtained with amyloid. Fuchsin stained the hyaline very deeply. Iodine coloured it light yellow. In the central region the hyaline mass was extremely tough, so that it was with difficulty disintegrated by firm pressure on the cover glass.

Liquor potassæ broke it up into masses and caused it to swell. Acetic and sulphuric acids were without action upon it.

Conclusions.—Reviewing briefly the clinical condition, there was first a severe constitutional condition of anæmia, and when this seemed to be improving the paralytic symptoms set in, and they were of the type of an acute ascending paralysis. The paralysis began in the lower limbs and then involved the hands and arms, but there it stopped: preceding the paralysis there were the ataxic movements, showing a probable lesion of the posterior columns. We had here to deal with a compound lesion involving both the grey matter and the white. The wasting of the limbs apart from other considerations compelled the reconsideration of the diagnosis of Landry's paralysis, and it was then considered to be a case of "Subacute diffused atrophic paralysis" ("*paralysie générale spinale antérieure subaigue*," *Duchenne*).

The *post-mortem* changes in regard to their sight corresponded to the clinical symptoms. The ataxia was explained by the lesion of the posterior column, and the motor atrophic paralysis by the almost complete destruction of all the ganglion cells along the whole course of the cord.

In regard to the nature of the lesion, it is probable that the previous condition of the blood when the patient had been suffering from profound anæmia was the prime factor at fault. The composition may have been altered and some albuminoid product produced. This formed inside the vessels and then exuded through the walls, rendering the latter homogeneous and hyaline. The substance then accumulated round the vessels in the cord and pia, and finally gained the lymphatic sheaths from which it came to act directly on the nervous elements of the cord, cells, and axis cylinders, causing complete atrophy and degeneration.

The distribution of the hyaline material was peculiar. In the cervical cord it was in enormous quantity, and had broken down, forming a rugged cavity, while below it got gradually *less* and *less* in the *posterior* columns, differing altogether from an ascending degeneration. This is probably another proof of its distribution by the blood vessels. The central mass, which was so apparent, seems to have been derived from filling up of the canal by hyaline, which had exuded from the central arteries of the cord.

For much help in the explanation of the complicated changes which were present in the cord, and for the unstinted use of his laboratory appliances, I desire to tender my best thanks to Professor D. J. Hamilton, of Aberdeen.

TREMOR IN GRAVES' DISEASE.

BY A. MAUDE.

THIS important symptom was first discovered by Charcot in 1856,[1] in the earliest French record of a case of this disease: Trousseau also noted excessive tremor in one instance. It did not escape the observation of Sir Morell Mackenzie in 1868,[2] while Dr. Hector Mackenzie[3] finds it noted in the records of St. Thomas' Hospital more than a dozen years ago.[4]

But on the whole this important factor in the recognition of the disease seems to have attracted little attention in England. Dr. Gowers' book is the only English text-book which mentions it, and in the discussion at the Ophthalmological Society in 1886, Dr. S. West, alone alluded to it.

Unquestionably it is a very constant symptom in Graves' disease, for I found it present in a typical form in six patients of a series of nine taken at random;[5] only two years ago, however, a patient was shown at a provincial medical society, and the tremors were ascribed to alcoholism.

The symptom has been very fully studied in France, chiefly by Charcot[6] and Marie, and the important monograph[7] of the latter embodies most of our knowledge on the subject.

[1] *Gaz. Med. de Paris*, No. 38.

[2] *Trans. Clin. Soc.*, 1868.

[3] *Lancet*, ii., 1890, 545.

[4] Russell. *Med. Times & Gaz.*, 1876, p., 250.

[5] A number of references in French literature are given by Marie. The more important are:—Chvostek, *Weiner Med. Presse*, 1870, Nos. 38, 39, 40, 42; Féréol, *Soc. Méd. des Hôp.*, Nov. 13, 1874; Delasiauve, *Soc. Méd. des Hôp.*, Nov. 27, 1874; Raynaud, *Thèse de Paris*, 1875; Rey., *Thèse de Paris*, 1877; Teissier, *Congrès de Clermont pour l'avancement des sciences*, 1876; Douglas, *New York Med. Journal*, 1879; Russell, *Med. Times & Gazette*, p. 250, 1876; Wynne Foot, *Dublin Journ. of Med. Sc.*, Nov., 1880; Nothnagel, *Wiener Allgemeine Zeit.*, p. 501, 1882.

St. Bartholomew's Reports, xxvii., 1891. Cases of Grave's disease, by A. Maude.

[6] See *Leçons du Mardi*, 1888-9, p. 231 (et sequit.).

[7] *Contributions à l'Etude et au diagnostic des formes frustes de la Maladie de Basedow*, Paris, 8vo.

I think it would not be too much to say that tremor is always present at some time or other in the course of the disease; even when we fail to find it on examination we shall learn that it occurs on excitement, or on any sustained muscular movement. In cases that begin rather suddenly, and settle down into an ill-marked condition, it has generally existed early, and does not last very long; but as a rule tremor is a late symptom. One patient of mine who had had goitre and exophthalmos for twenty years, had never noticed the tremor till a year before she came under my charge; and in all my cases except two it has been long after the establishment of other cardinal signs that it appeared. In one instance the patient, after some years of futile treatment by drugs, has improved very greatly under the continued use of a Leiters tube to the epigastrium; tremor has been present in a slight degree for a year or two, but since the general symptoms have improved the tremor has become far more persistent and marked.

The degree varies so much that in many cases the patient is quite unaware of it, and the condition is generally only noticed at first by the subjects of it because it interferes with delicate movements of the fingers, such as writing or sewing; but it may become so severe as to cause her to drop cups and plates, as in my patient's case, or even to prevent walking,[1] or preclude the physician from feeling the pulse.[2]

My experience confirms the observation that the movements are as a rule very small in amplitude, strikingly regular, and are not dependent on voluntary motion.

The vibration is most marked in the hands, and may in very slightly marked cases be confined to them. The other muscles most affected are the pectorals, the serratus magnus and the erector spinæ. But if the tremor is well marked in any group of muscles it is sure to be general, over the whole body, to some degree. I have seen one lady who had never noticed it in her hands, where it was slight, but had noticed it in the muscles of the back; it was confined to these two muscular groups.

Tremor is usually symmetrical, but not necessarily; and

[1] Charcot. [2] Russell.

has been known to be unilateral, when the goitre or exophthalmos are also unilateral.[1] In cases where it is well marked, the patient is in a state of perpetual vibration, sitting or standing. It is then much marked in repose, and only slightly exaggerated during movement.[2] But though muscular repose does not necessarily free the patient from tremor, mental repose does so to a great extent. It is always increased by agitation or "nervousness": it is produced or increased by hurried muscular action, or by any attempt to perform a movement carefully or accurately, especially if the patient is aware of her tremulous condition.

It is easy to ascertain the existence of even slight tremor by placing the hands on the patient's shoulders when she stands up. In this position the vibrations are, in well-marked cases, best felt in the vertical direction, but even in slight cases, where no vertical movement is felt, a small lateral oscillation can often be seen, if not felt.

On stripping the subject we may perceive that the muscles of the extremities and trunk are in a state of continual fibrillary movement, "that the whole surface of the body is the seat of a kind of general palpitation of a most curious character." (Marie.)

The tremor may be so exaggerated as to affect the respiratory muscles, producing a jerking laryngeal sound audible at some distance.[3] To study this condition carefully, make the patient stretch out her arms, stripped, with the hands lightly extended. The whole limb is then seen to be affected by rapid contractions of the muscles, the amplitude of which varies in different subjects. The movement is usually fine, and is always quite regular and constant. The oscillations are most easily visible in the hands, and they affect the hand as a whole, being produced in the flexor and extensor groups of the forearm. The fingers have little individual movement, and the interossei especially have no share in it. The wrist is the joint most affected by the oscillations, and probably the fact that the flexion and extension movements are more marked than the supination

[1] Gowers. [2] Féréol. [3] Charcot, *loc. cit.*

and pronation is due to the greater ease with which the wrist moves in the former plane, than to any election of groups of muscles in the forearm on the part of the tremor. The oscillations are more visible in the hands, not because the hands are farther from the point of support of the limb, but because the muscles of the forearm are affected most.

This "flexor tremor" is also characteristic of alcoholism, but at the same time in alcoholism there is distinctly more contraction of the interossei and consequent lateral deviation of the fingers.

In the same way the movements of the lower limbs may be studied. Let the patient stand with the foot extended, just pressing the floor with the toes; the vibrations are then usually marked. Then let her sit with the knees at a right angle, and the toes touching the floor, and we shall find in a well-marked instance, the same sort of vibration as in "ankle-clonus" (Fuss phœnomen). In this connection I examined the patellar reflexes in five cases, and found them absent in four, and feeble in the fifth. Tremor is usually constant in any one patient for a long period, but it has terms of exacerbation, just as have all the other symptoms; so that in many cases the habitual or constant tremor passes unnoticed by the patient, but the exacerbations are observed.

I have not succeeded in finding any direct relation between the increase of tremor and increase of other symptoms.

Marie has obtained mechanical tracings of this tremor, and gives us an excellent account of them in his monograph; and to his work I am entirely indebted for knowledge on this part of the subject.

Registration of the vibrations was obtained by two methods. First, by a modification of Marey's tambour, applied to the dorsal aspect of the metacarpus; secondly, by means of an india-rubber ball or "pear" grasped in the patient's fist (like an enema syringe) not tightly enough to compress it. Of course, in both cases the tambour and "pear" are connected with an ordinary recording apparatus, furnished with a tuning fork to record the rate.

Marie and Boudet also tried to obtain tracings by means of Rosapelly's apparatus; but the instrument only gave the frequency of the vibrations, without any record of their amplitude.

In all comparative experiments of course the same mechanism, pressure and tuning fork must be used.

From the tracings we learn, first as to the *rapidity* of the oscillations, that they are strikingly regular in rhythm; the interval between the crests of the successive waves varies very little; in fact the tracings are almost as regular as those of a tuning fork.

In eleven cases, in which tracings were taken by Marie, the rate of movement was almost the same in all, varying only from $9\frac{1}{2}$ to 8 oscillations per second, with a mean for all cases of $8\frac{1}{2}$. As to amplitude, in the vertical direction the oscillations present anything but regularity. If we examine tracing 1 of Marie, (p. 20), we see that the waves increase gradually for a certain number and decrease again. The tracing presents a series of roughly fusiform enlargements, described as nodes (nœuds) by M. Fernet in his monograph.

These variations in amplitude are very irregular, and no explanation has yet been offered as to their causation, but they certainly have no correlation with the movements of respiration or circulation. A distinct correlation is however found to exist between the increased waves and the occurrence of cough; an attack of cough produces a greater amplitude of vibration without disturbing the rate. This change is probably merely due to the shock of the chest wall, produced by the cough, being transmitted through the shoulders and arm to the hands.

In reply to the question whether there is any direct relation between the degree of tremor and the intensity of other symptoms, my own impression is strong that there is none. Not only, as I have said above, may it become most pronounced as other symptoms are improving, but it is more constant, in individuals who present it, than any of the other (notoriously inconstant) symptoms of the disease.

It has also no relation to a general emotional (or "nervous") state, as hysterical tremor often has.

When we compare the tracings produced in Graves' disease with those produced by other forms of tremor, the difference is instructive. Such other forms are senile tremor, paralysis agitans, general paralysis, disseminated sclerosis, hysteria, and poisoning by alcohol, mercury, lead or arsenic.

The tracings obtained in Graves' disease are exactly

similar in time and form to those produced in animals after excision of the thyroid gland.[1]

Mr. Horsley also drew attention to the fact that the tremor wave in paralysis agitans is a compound wave, a summation of two separate waves, as the larger convulsive tremor waves in myxœdema are summations of smaller ones.

The correspondence of the tremors in myxœdema and exophthalmic goitre afford some evidence of the seat of production of the tremor; for Schiff had shown that the tremor in cachexia strumipriva was of central origin, and Horsley confirmed the observation, while he carried the investigation further, for he removed in one case the centre in the cortex for the upper limb; the result was complete brachioplegia, but no interruption of the tremors; thus proving that the tremor originated not in the cortex but in some lower centre, either in the basal ganglia, the bulb, or the spinal cord; and support is given to this assumption by the fact that at first voluntary effort abolishes the tremor, that the inhalation of ether increases it, and that it disappears on reflex irritation.

Moreover the rate of vibration is 8 to 10 per second at first, and later some multiple of these numbers, which is the rate determined by Loven for the spinal cord in frogs, and probably also the rate for the spinal cord in man (Horsley).

Mr. Horsley, however, admits that none of these experiments disprove the possibility of the tremors being idiomuscular.

In the disease we are studying we have seen the rate of vibration is very constant, 8 or 9 per second; in senile tremor it is much more slow, $4\frac{1}{2}$ to $5\frac{1}{2}$ (Marie); in no case above 6 per second. In paralysis agitans the rate varies considerably in different cases; it may simulate disseminated sclerosis in its slow rate and its irregularity. Charcot gives an average of 3 to 6 oscillations per second, and Marie an average of 5. On the other hand it is quite exceptional for the movements of insular sclerosis to be as fine and regular as in Graves' disease.

The movement in Graves' disease and mercurial poisoning are shown by Charcot[2] to differ only in rate, and in the fact that in the former affection they are much more continuous and regular, and are not increased, as a rule, by intentional muscular action.

In general paralysis and alcoholism the rate of vibration may be much the same as in Graves' disease, but tracings

[1] Brown Lectures, 1885. [2] *Leçons du Mardi*, 1, p. 378.

show that while the general line in the latter is regular and straight, that produced in the other two affections is much more wavy and jerky. Moreover in Graves' disease the intrinsic muscles of the hand and tongue escape, while they are particularly affected by alcohol poisoning.

Tremor in lead poisoning is so rare and varies so much in character that it is impossible, as yet, to draw distinctions on this head.

An important feature which separates that of Graves' disease from tremor due to metallic poisoning is the entire escape of the muscles of the head, neck, lips and tongue.

I have not sought to press the diagnosis of this form of tremor from others, but merely by indicating what I believe to be its salient features, to leave to the cultivated neurologist its differentiation from other forms better known.

The affinities of this tremor are rather to purely "asthenic" trembling, such as follows in many subjects an acute illness, or to the tremor of old age.

From hysterical it is separated broadly by its great regularity in rhythm, but, of course, so-called hysterical trembling may resemble that of Graves' disease, as it will occasionally imitate that of any other neurosis.

The points on which I would lay stress, are :—

(*a*) The great frequency of it in cases of exophthalmic goitre.

(*b*) Its constancy when it does exist.

(*c*) Its constant character.

It may be observed that I make no mention of chorea in this neurosis. The condition has been noted by observers fully capable of differentiating true choreic movements, but there is no evidence as to whether it exists or not side by side with the ordinary trembling.

We ought not to lay too much stress, except for diagnostic purposes, on differences in tremor in various diseases. We know nothing about the pathogeny of tremor, or of the seat of the lesion, and it may well be that what appear to be distinct forms of trembling owe their difference to the fact that certain muscles are affected rather than to the fact of different causation.

REMARKS ON THE PATHOLOGY OF SO-CALLED PACHYMENINGITIS INTERNA HÆMORRHAGICA.[1]

BY J. WIGLESWORTH, M.D.LOND., M.R.C.P.

Lecturer on Mental Diseases University College, Liverpool, and Examiner Victoria University.

It would be foreign to my purpose to-night to enter upon a detailed account of the affection commonly known as *Pachymeningitis interna hæmorrhagica*, my object rather being to offer a few remarks in favour of the hæmorrhagic origin of the affection, in opposition to the view which ascribes the phenomena met with to an inflammatory process, which latter view, if we are to judge from descriptions given in text-books, appears to be still the prevailing one.

As is well-known the hæmorrhagic theory is not a new one; it is in fact the older one of the two, and was advocated by Prescott Hewett and others, before Virchow lent the weight of his authority to the inflammatory doctrine, and it is mainly to the writings of that eminent pathologist that we are indebted for the predominance of the inflammatory theory at one time almost universally accepted.

It is unnecessary for me to quote the usual description given in text-books when treating of this affection; the formation of an inflammatory film upon the inner surface of the dura mater—the organization of this—the formation of similar films superimposed one over the other—the rupture of newly-formed vessels—and so on. All these descriptions start with the assumption that the primary thing is an inflammatory exudation upon the inner surface

[1] Read at a meeting of the Neurological Society, May, 1892.

of the dura-mater, and that the hæmorrhagic element met with is a secondary result of the rupture of newly-formed vessels in this inflammatory membrane.

I take the exactly opposite view to this, and contend that the membranes found adherent to the inner surface of the dura-mater, be they simple or laminated, small or large, filmy or thick, hæmorrhagic or fibrinous, are all the direct result of hæmorrhagic effusions into the sub-dural space (arachnoid cavity); and that any inflammatory changes which may be met with in the dura-mater, are purely secondary and due to the irritation set up by the effused blood.

I may briefly sum up the chief arguments in favour of this view as follows:—

If the membrane were the primary thing, and the hæmorrhage secondary, although we might get the membrane without the blood, we could not get the blood without the membrane.

But as a matter of fact cases are continually met with—I have seen many such—in which effusions of blood are found in the sub-dural space without the presence of any trace of membrane upon the inner aspect of the dura-mater. Sometimes the effusion is a copious one, at other times a little recent blood or clot is found smeared over the upper surface of the arachnoid, or a recent clot may be met with loosely adherent to the inner surface of the dura, just stuck on as it were; or all these conditions may be combined. Of course it may be argued that these cases should not be included in the same category as those in which a distinct membrane is present; but the two things occur under such exactly similar sets of conditions, and there is such a very gradual transition from one to the other, that the conclusion seems forced upon us that they are merely stages in the same pathological process.

Again, if the membrane were the result of a primary inflammatory exudation from the vessels of the dura-mater we should surely find some evidence of inflammatory changes in this structure itself. But, as a matter of fact, such changes are for the most part conspicuous by their absence.

This is so at least in all quite recent cases. If we detach the membrane from the inner surface of the dura-mater, to which it is always but loosely adherent, we find the endothelial surface of this latter membrane smooth and shining and exhibiting no sign of departure from health. There is no thickening, no roughening, no undue vascularity. Later on, as the new membrane tends to become organized, vascular adhesions form between it and the dura-mater, and on the separation of these, which can always be readily effected, the inner surface of the dura may be found a little roughened and at times a little injected, and the dura itself may perhaps be a little thickened. But, I repeat, even these changes, slight though they be, are not found in the recent cases, and I regard them purely as secondary results of the irritation set up by the effused blood, and as quite comparable to the changes which take place in the walls of a vessel within which coagulation has occurred.

The structure of the newly formed membrane itself is entirely in harmony with the supposition of its hæmorrhagic origin; for if recent it is found to consist of a meshwork of fibrin in which are entangled red and white corpuscles; whilst in the older cases, when capillary vessels have been formed in it, bands of imperfectly formed fibrous tissue may be seen running parallel with one another containing in their interstices collections of red blood globules, or it may be simply the remains of these in the form of little heaps of hæmatoidin granules; the whole in fact having much the appearance of an organizing or organized thrombus.

The age at which this affection is most frequently met with is distinctly that of advancing life with its concomitant arterial degeneration and tendency to hæmorrhage. Out of fifty-four cases observed by myself, which I have analysed with reference to this point, the average age was 51.61; the mean age of the asylum population from which the cases were drawn being about 43. When occurring in young persons it is almost always in association with general paralysis, in which disease it is common to meet with degenerative changes in the small vessels of the pia-mater and cortex cerebri.

It is worth noting that the affection is by no means always bilateral, although it is so in the majority of cases. Thus out of fifty-four cases, twenty were entirely unilateral and thirty-four bilateral. One would have anticipated that had the affection been an inflammatory one it would have affected more uniformly both sides of the cranium.

The rarity with which the affection occurs in the cerebellar fossæ is also of interest. Whilst it is not uncommon to meet with a hæmorrhagic membrane on the upper surface of the tentorium cerebelli, it is very rare to meet with one beneath it. I do not think I have ever seen an instance of it. On the inflammatory theory this is difficult to account for; but the mechanical obstacle which the tentorium must present to the gravitation of blood into this region, supplies a ready explanation of its exemption.

The conditions under which this so-called pachymeningitis is met with are of importance in determining its pathology. Unquestionably the immense majority of cases are met with amongst the insane, and hence it is chiefly in asylum practice that it comes under notice. Next in frequency it occurs in the subjects of chronic alcoholism, a condition indeed closely allied to insanity. Its occurrence under other circumstances appears to be very uncommon, if we are to judge from published records; and on this point I am anxious to hear the experience of others. Furthermore, it is not in the recent acute cases of insanity that it is commonly found; although it occasionally so occurs, it is nevertheless comparatively rarely met with except when the insanity has been of some duration. General paralysis is the disease in which it most commonly occurs, about half the total cases of pachymeningitis having this association; but no form of insanity is exempt, the different forms of dementia furnishing a good proportion. In other words the affection is associated in overwhelming proportion with *brain degeneration and atrophy*. And this leads me to what I consider to be the main factor in the production of the disease, namely, the loss of support experienced by the meningeal vessels in the degenerating and wasting brain, assisted no doubt by the localised or general congestions of

the meninges which are so liable to occur in all forms of insanity, especially in general paralysis. Probably the source of the hæmorrhage varies in different cases; it unquestionably at times comes from the small vessels of the pia-mater occupying the summits of the gyri, for I have seen cases in which a recent clot, or a hæmorrhagic membrane, has been associated with a small sub-arachnoid hæmorrhage in this situation, and have been able to trace the rupture in the arachnoid through which the blood has been effused into the subdural space. That the blood does not diffuse itself more extensively throughout the subarachnoid space is doubtless due to the fact that in many cases of insanity, especially in general paralysis, there is a more or less intimate union between the pia-mater and arachnoid, so that in bursting through this latter membrane into the subdural space the blood would be simply following the lines of least resistance.

A further fact of some significance is that as a rule the affection does not declare itself by symptoms, the pachymeningitic membrane being discovered after death in cases in which its presence had not been suspected during life. Doubtless this is in part, perhaps mainly, due to the fact that the affection usually occurs in insane persons in whom symptoms of all kinds are masked. But even demented patients are liable to be disturbed by affections producing brain irritation and pressure, and I cannot but think that the comparative rarity of symptoms is another argument in favour of the compensatory character of the affection, the inference being that in the majority of cases the effused blood does little or no more than fill up the space left by the wasting brain. In a minority of cases, however, the hæmorrhagic effusion, once started, is not so readily limited, but becoming copious produces signs of irritation and pressure, declaring itself by such symptoms as convulsions, paralysis, &c. I could narrate several such cases, in some of which a copious effusion proved rapidly fatal, whilst in others, the patients having lived a variable time after the onset of symptoms indicative of such an effusion, there was found after death a hæmorrhagic or fibrinous membrane adherent to the under surface of the dura-mater.

A word or two with respect to the lamination which many of these pachymeningitic membranes present. Doubtless the appearance of successive layers has had much to do with the notion of an inflammatory process, but I fail to see how this condition favours the idea of inflammation as opposed to hæmorrhage. I think that without doubt this laminated appearance is due at times to fresh hæmorrhagic films being superimposed upon those of older date which have already become more or less organized, the same conditions which produced the first effusion continuing in operation. But I have little doubt that a single large hæmorrhage is capable of producing in time a laminated membrane owing to the changes which take place in the clot. One of the specimens exhibited from a case already published illustrates this. Here the hæmorrhagic membrane which was clearly occasioned by a copious hæmorrhage seven days before death, showed when fresh a dark centre bounded by pale lines and a tendency for the fibrin to split into layers parallel to the surface—clearly an incipient lamination. It is not, however, to be denied that rupture of newly-formed vessels, in an organizing membrane of this description occurs at times, producing fresh hæmorrhages. I show a figure and drawing of such a case in which a small recent clot is seen lying between two thin layers of membrane, producing when fresh something of the appearance of a cyst—hence one of the names, "Arachnoid Cyst," under which this affection has been described. The hæmorrhages thus produced, however, appear to be mostly of very small dimensions.

In bringing before you these few facts and arguments in favour of the hæmorrhagic origin of the affection under consideration I do not wish to be understood to deny the existence of a true cerebral pachymeningitis.

All I wish to contend for is, that the condition which usually passes under that name has no right to be called by such a title; and that it would be better to discard it altogether, and adopt one more in accordance with pathological facts.

Clinical Cases.

A CASE ILLUSTRATING KINÆSTHESIS.[1]

BY WILLIAM B. RANSOM, M.A., M.D., M.R.C.P.

Physician to the General Hospital, Nottingham; late Fellow of Trinity College, Cambridge.

THE following case of epilepsy, treated by trephining, afforded opportunities for observations and experiments such as have hitherto been few in man, and gave information on the subjective side which cannot be obtained from experiments on animals. Although therefore in some respects the results are less rigorously accurate than those of the physiological laboratory, yet I venture to think they shed some light on the mode of action of those areas of the cerebral cortex associated with voluntary motion, which by a majority of physiologists have been called simply "motor centres," but which Dr. Bastian considers to act more on the sensory side of the series of cerebral processes, and has called "Kinæsthetic."

It would be superfluous for me here to set forth the opposing arguments of the two schools, and I shall simply record briefly the aspects of this case which bear upon the problem.

History of Case.—T. F., a young man of 19, came into hospital under my care in April, 1891, complaining of fits.

Six years previously he had been knocked down and rendered momentarily unconscious. He walked home, however, apparently not much the worse, but next night had his first fit. During the next nine months the fits gradually increased in number and severity. Each fit began with tingling in the left thumb, which

[1] Paper read August 2nd, 1892, at the International Congress for Experimental Psychology, London.

was then the seat of spasm, which spread up the arm to the shoulder and face. He then usually lost consciousness. Occasionally he seems to have had the tingling sensation without subsequent spasm.

A year after the accident he had a very serious illness, probably meningitis, which left him blind. He recovered from this, but the fits continued, though with less severity.

A curious point is that about a year after this illness he began sometimes to have a visual aura preceding that in the thumb. This consisted in seeing "queer objects" and bright lights.

Condition in April, 1891.—Patient is a well-grown young man, with no motor defect except in the left hand. General sensation is normal except there. The sense of smell is absent, and he can at most perceive the difference between a dark room and bright sunlight. The optic discs are atrophied. There is no depression or scar on the head.

Left Hand.—There is some weakness of the muscles of the left hand: right grip = 160, left = 75. No wasting of muscles.

Tactile sensation is dulled below the wrist, particularly on the thumb, and the power of localisation is bad, two points being merged into one at a much greater distance than on the right side. There is partial analgesia. The muscular sense is markedly deficient. He fails to make out the position of his fingers or thumb, or to distinguish weights which are readily known as different by the right hand.

He cannot read his raised type with the left hand. All these deficiencies are more marked in the thumb.

Operation.—As treatment by bromides failed to relieve, I asked my colleague, Mr. A. R. Anderson, to operate.[1] This he did by trephining at a point $2\frac{1}{2}$ inches down the right rolandic line. Subsequently another disc of bone was removed, and a surface of brain about 2 inches by 1 inch exposed. A circular patch of this area, about 1 inch in diameter, was found to be depressed by a cystic accumulation of fluid between the pia and the thickened dura mater. Moderate faradization of this depression caused flexion of thumb and fingers; when the current was increased the contraction spread up the arm to shoulder, left side of face and left leg, and then a general fit ensued. The application of the electrodes to the healthy brain at the lower margin of the depressed area caused twitching of the left side of the face; when applied above the depression the left shoulder contracted.

[1] Mr. Anderson will publish the surgical aspects of the case elsewhere.

I merely mention these details to show the topographical agreement between man and monkey.

The thickened dura mater having been cut away, the bone discs were replaced and the wound closed. The patient made an excellent recovery. For a time he had fewer fits, but eventually they recurred and he began to have considerable pain in his head. It was accordingly decided to remove the discs of bone, and this Mr. Anderson did on March 29th, 1892, removing at the same time adhesions to the cortex. A moderate faradic current applied at the operation to the centre of the depressed area caused contraction of the thumb only. The patient again recovered readily, and left the hospital a month after the operation. Since then he has had no pain, very few fits, and those slight ones. I have carefully watched his progress, and have been able, with his full consent, to study the effect of faradizing the cortex through the gap in the bone without the sensorium being obscured by an anæsthetic.

The following method was adopted:—

The skin having been shaved and cleansed antiseptically, is dried with alcohol, and a little cocain is injected into the scalp to prevent the excitation of the cutaneous nerves becoming too painful. (The cocain may be omitted, but the skin sensations are, in that case, apt to be unpleasant.) The electrodes, two strong sharp needles, mounted at a distance of $\frac{1}{4}$ inch apart, are then thrust through the scalp into the trephine hole for a depth of half-an-inch.

The first onset of even a weak current may be somewhat unpleasant to the skin, but this soon wears off, and even strong currents can be borne.

As the electrodes at a depth of half-an-inch are probably not in actual contact with the cortex, moderately strong currents are needed to get definite results, but that diffusion does not take place to a wide extent is proved by the fact that the insertion of the electrodes in different parts of the scalp over the trephine hole caused distinct groups of muscles to contract. Thus movements of the elbow were caused when the needles were in the upper part, of the fingers when lower down; and I succeeded once in getting flexion of the fingers when the needles were in one part of the hole, and extension when they were removed half-an-inch away.

The following changes have occurred in the hand since the first operation. Motor power is somewhat better than before, but still rather weak; there is no analgesia, and tactile sensation is

distinctly improved, being now nearly as acute as on the other side, except on the ring and little finger. Muscular sense is similarly better, being also most deficient on the ring and little fingers. (It is curious that whereas before the operation the thumb was distinctly worse than the fingers, it has recovered to a larger extent.)

In making an experiment, tactile sensation, the muscular sense and motor power were tested before and after stimulation, and in some cases muscular sense was tested during the stimulation.

The following results were obtained :—

(1) It was possible, as already mentioned, by this method to cause contraction of different groups of muscles of the arm and hand.

(2) The first effect of a moderate current was to produce *a tingling sensation* in the part which would contract when the strength of current was increased. After repeated excitations with a given strength of current the contraction might cease to be produced, and only the sensation occur. The sensation might outlast the stimulus.

(3) After a strong induced contraction, voluntary motor power was weakened.

(4) While muscles were contracting under the influence of the current, this muscular sense was improved, and this improvement lasted a few minutes after the application of the current.

(5) No effect was observed on tactile sensation.

The above are the results which bear chiefly on the doctrine of kinæsthesia, but certain other points of interest may also be mentioned :—

The contractions caused by such stimulation might be limited to one finger or the thumb, or might affect a large group of muscles.

Thus in one experiment the first effect was a sensation in the fingers, then tonic closing of fist, then tonic flexion of elbow, and then clonic spasm of the whole arm. The spasm did not spread, and consciousness was not lost.

In another experiment the same result occurred, but in this, although the needles were removed when the spasm reached the shoulder, it spread to the leg and face, which

latter became congested. He never lost consciousness, but was able to get up off the chair and lie on the floor as a precautionary measure. He said "that was just like a real fit." As a rule, however, only moderate contractions, strictly limited to a few muscles, were obtained, so that it is probable that in the above two instances the hyperæsthetic epileptogenous focus was excited.

Another very remarkable phenomenon sometimes ocurring was that during stimulation with quite a moderate current he would see a light, blue or white, mostly in the left eye, just as he frequently has done at the beginning of a fit. Possibly this visual aura and visual sensation produced by excitation of the arm centre may be connected with some pathological condition produced by the serious illness in which he went blind six years ago.

Great caution is needful in drawing conclusions from these observations, but I venture to think they go to show :—

(*a*) That depression of the activity of the cortical so-called "motor" centres is associated with the following changes in the corresponding limb or part of limb :—

(1) Diminution of motor power.

(2) ,, muscular sense.

(3) ,, to a less extent of general sensation.

(*b*) That exaltation of their activity, even when artificially produced by the interrupted current, leads to the following changes in the corresponding limb :—

(1) A vague sensation.

(2) Increase of muscular sense.

(3) Muscular contraction.

These conclusions I further think are not readily explicable on the theory of motor centres with which Dr. Ferrier has especially associated his name, but indicate a close connection with the afferent as well as the efferent series of cerebral processes, such as is postulated in the Kinæsthetic theory of Dr. Bastian. It would perhaps be fortunate if we could drop the term "centre" altogether, inasmuch as it implies the idea of an end-organ, remembering that whether viewed from the physical or psychical

aspects, vital actions are really reactions with a complicating time factor, and that to look upon these shunting places of nervous currents from the ingoing or outgoing side alone must always obscure half the truth.

NOTE.—At the meeting at which this paper was read Prof. Schäfer made the perfectly valid criticism that the sensation produced in the hand as the first result of electrically stimulating the "motor area" of the cortex might be due to vaso-motor changes in the hand, which changes were imperceptible to the observer. I am quite aware that it is impossible to prove the non-existence of unperceived processes (such as vascular changes or small muscular contractions), due to efferent impulses going from cortex to hand, which might be the cause of the sensation; but we cannot thus account for the complementary fact, viz.—diminution in tactile and muscular sense during depression of the activity of the cortical area.

TUMOUR OF CENTRUM OVALE, CAUSING PARALYSIS OF MOTION AND MUSCULAR SENSE; OPERATION; DEATH.

BY LANDON CARTER GRAY, M.D., NEW YORK.

I DO not deem it necessary to recapitulate all that has been written of late years concerning localization of the muscular sense, or to more than refer to the compilations by Dana and others. I think that the following case is really the first record, in the happy precision of the localisation and the short duration of the symptoms. It seems to me to settle beyond a doubt the question that lesion of the ascending parietal at least, can cause a loss of the muscular sense without impairment of the other senses.

On November 1st, 1890, Dr. I. H. White, of Richmond, Va., brought to my office a gentleman aged thirty-eight years, of whom the following history was given :—

Some fourteen days before, his right arm and leg had gradually become weak, the onset having been first in the foot, and then, a few days afterwards, in the arm. For about the same time he had suffered from headaches, worse in the morning, at first at the vertex, occasionally in the left side of the head, and on the morning of my examination across the brow. He had also, for about the same length of time, suffered slightly from insomnia, so that he could obtain only about four to five hours' sleep. Several years before this patient had what Dr. White supposes was Menière's disease. Some time before my examination an angioma had been removed by the doctor from the region of the knee by means of caustics. About a year before a hard growth had appeared in Scarpa's triangle, and now occupies the whole of this anatomical area. There has been great domestic trouble, about which the patient has been much worried. The patient has always stuttered, and this speech-defect does not now seem to be worse, and he has had no other defect of speech than this. There was a gonorrhœa twenty years ago, and following it possibly, Dr.

White thinks, a slight stricture. There has never been any discharge of pus from the ear, but there would seem to have been, at the time of the Menière's disease, some acute aural trouble, either of the labyrinth or the middle ear, and there is now deafness upon this side. There is no rheumatic or cardiac trouble, and there has never been any convulsion or loss of consciousness. The iodide had been administered in doses of fifteen to twenty grains, but invariably disagreed so greatly with the patient's stomach that it could not be pushed higher, or, indeed, continued.

Upon examining the patient myself, I found that there was a considerable motor paralysis of the right leg, so that the patient walked with great difficulty, and that there was also a slight motor paralysis of the right upper extremity, so that the hand-grasp was distinctly more feeble than upon the other side. In the foot the sensations of tact and pain were very slightly impaired, whilst the sensations of temperature were perfect. Tact was tested by means of the æsthesiometer, and by the differences between a rough cloth and the finger, and by the perception of objects held in the hand. The sense of pain was tested by means of a needle. The sensation of temperature was tested by means of three tumblers, one containing hot, one lukewarm, and the other cold water, and the patient promptly detected the difference in these by touching the outside of the glass with his fingers. But the muscular sense was so distinctly impaired that the patient was unable to tell which finger I took hold of, or what I did with it, or to tell me the nature of the muscular movements which I imprinted upon the foot as a whole. In the right hand the sensations of tact, pain, temperature, and the muscular sense were absolutely unimpaired. The cremaster reflex was very sluggish on both sides. The tongue pointed straight. The naso-labial folds were strong and equal. The right knee-jerk was slightly exaggerated and somewhat spasmodic, whilst on the left it was normal. I could detect no mental impairment whatsoever, although I ranged over a wide field of discussion involving judgment, perception, memory and temper. There was great deafness of one ear, seemingly due to an old otitis media. Careful ophthalmoscopic examination disclosed nothing abnormal about the discs. A detailed examination of the muscles of the eyes, eyelids, abdomen, and of all the muscles of the opposite side of the body, of the abdominal reflexes, and the tendon reflexes of the upper extremities, of the other ear, of taste, smell, and sight, disclosed nothing abnormal. I made a diagnosis of a tumour of the left centrum ovale. I believed there was a tumour because of

the presence of one in Scarpa's triangle, as well as because of the history of the precedent angioma. I believed that the tumour was subcortical because of the lack of a convulsion, together with the absolute mental integrity. I stated to the patient that I thought his paralysis would soon involve the arm and progress in the leg, advised him to go home and settle his affairs, advised his physician to administer as large doses of iodide and mercury as the patient could stand, and then, if the disease progressed, to have the patient trephined.

On November 13th the patient was again brought from Richmond by Dr. White. His leg was now completely paralysed, as was also the upper extremity, so that the patient could only make a slight movement of the fingers. The sensations of tact, pain, temperature, and the muscular sense were impaired to the same extent that they had been in the examination thirteen days before. In the right hand, however, the sensations of tact and pain had now become very slightly impaired, but the latter was not at all retarded; the temperature sensations were perfect, but the muscular sense was distinctly impaired, though not greatly so, the patient being unable to tell as to which one of the fingers was touched, or what movements were impressed upon any one of them, although he could speak decidedly when the whole hand or the arm or forearm were moved. The tongue still pointed straight, and the naso-labial folds were still strong and equal. The cremaster reflex was still very sluggish. In all other essential details, however, the patient was unaltered, except that his headache had now become intense, and he complained bitterly of it. This headache was not now confined to any particular portion of the head, although he seemed to think that it was greater over the brow. Although the patient was very somnolent, his intelligence was perfect, so that he put and answered questions with perfect intelligence, although slowly, and he gave me an accurate account of how he had willed his property and otherwise disposed of his affairs. In the last few days he had complained of roaring noises in the deaf ear. Since I last saw him he had had no convulsions and no vomiting. The left eyeball seemed slightly more protruberant than the right, but there was no ptosis nor strabismus.

On November 14th, at 2 p.m., he was operated on by Dr. John A. Wyeth; Drs. I. H. White, E. F. Ayres, J. F. Wynn, and myself being present. The skull was unusually eburnated, and it took an unusual time to cut through it. Sufficient of it was removed to lay bare the ascending frontal, the ascending parietal,

the bases of the second and third frontal, and about the anterior third of the first and second parietal convolutions. The dura bulged and was very tense, although it seemed to be normal. The pia was normal and not at all injected. When the membranes had been removed, the cerebral substance bulged markedly. As careful an examination was made of the cortex and subjacent white matter as it was possible to do by means of palpation and puncturing with a long needle, and this needle was thrust as far down as the ventricles in several different places. But no sign of a neoplasm was felt or seen. The patient stood the operation remarkably well, but upon recovering from the effects of the ether remained comatose, except for a few moments, when he said that his headache was gone. The temperature ranged at about 100° until about an hour before death, when it rose to 108°. He died the morning of November 17th. The autopsy was made about twelve hours after death by Dr. Dawbarn, there being present Drs. John A. Wyeth, E. A. Ayres, E. G. Mason, W. B. Pritchard, and myself. A slight pachymeningitis, evidently of long standing, was found over the paracentral lobule, but the pia and cortical substance were seemingly healthy. The calvarium was found to be unusually thick, and so marked was this thickness at the apex of the petrous portion of the temporal bone as to constitute a hyperostosis, most noticeable on the right side. There was no periosteal adhesions of the dura. At the site of the operation there were inflammatory changes, although these were slight. There were numerous *puncta hæmorrhagica* in the white substance, corresponding to the punctures that had been made during the operation. The cerebrum and the cerebellum were removed from the skull, placed upon a flat dish, and sections of the cerebrum were made from side to side vertically across the cerebrum, each of these extending in depth from the cortex through to the base. The first section was made about half a centimetre in front of the mid-point of the upper or first frontal convolution; the second section was made midway between the first section and the anterior border of the ascending frontal convolution; the third section was at the anterior border of the ascending frontal convolution, and the fourth section was at the posterior border of the ascending parietal convolution. Other sections were made behind this last, of course, of which it would be irrelevant to speak. Even by these different sections no neoplasm was discovered. I then proceeded to minutely examine each one of these sections, first by palpation, and then by the scalpel. In the first three sections nothing was discovered by these methods of examination.

In the fourth section nothing was detected by palpation, but very slight manipulation with the handle of the scalpel disclosed, close to the posterior border of the fourth section, a neoplasm about the size of a hickory nut and so soft in consistence that it was not distinguishable by palpation from the surrounding cerebral substance. It was found to have a cystic degeneration in the centre, and around the tumour there was no microscopical evidence of cellular changes in the white matter. *It lay, as will be seen, in the ascending parietal convolution, rather nearer the posterior border of this convolution, and was situated about one quarter of an inch beneath the surface of the convolution, at about the junction of the upper and middle third.* Upon section it was found that the tumour had been penetrated by one of the exploratory needles. A microscopical examination, kindly made by Dr. Beach, showed that this neoplasm was a round-celled sarcoma of the melanotic variety.

ANOTHER CASE OF ANÆSTHESIA DUE TO LESION OF THE GYRUS FORNICATUS.

BY THOMAS SAVILL, M.D.LOND., D.P.H.CAMB.

MUCH difficulty surrounds the subject of the localisation of the cerebral centre for tactile sensation. In animals it is purely a matter of inference whether the animal has lost the power of feeling when he is touched or pricked, and in man it is necessary that the mental faculties shall be, at least to some extent, retained. Frequently the anæsthesia, if present, is so mixed with other phenomena, and attended by clouding of the intellect, that it is most difficult to come to a conclusion; no apology therefore is needed for placing the following case on record.

Some observers have placed the area for common sensation in the central region of the cortex, that part beneath the parietal bone, corresponding very nearly with the motor area, this, however, is now considered doubtful.

Ferrier was the first and for a long time the only investigator[1] who showed that lesions of the gyrus hippocampi caused anæsthesia of the opposite side.

The laboratory experiments of Horsley and Schafer[2] localise the centre for painful and tactile sensations also in the gyrus fornicatus, the convolution adjacent to the corpus callosum, on the median aspect of the hemisphere. Thus it would seem that the centre for sensation is located in the whole of the falciform lobe, and not exclusively in either the gyrus hippocampi or gyrus fornicatus.

In the autumn number of BRAIN, 1891,[3] I have recorded a case containing pathological evidence in support of the

[1] "Functions of the Brain," 1st edition, 1876.
[2] *Transactions of the Royal Society*, Vol. 179 (1888), B., pp. 1-45.
[3] BRAIN, Part LV., 1891, p. 270.

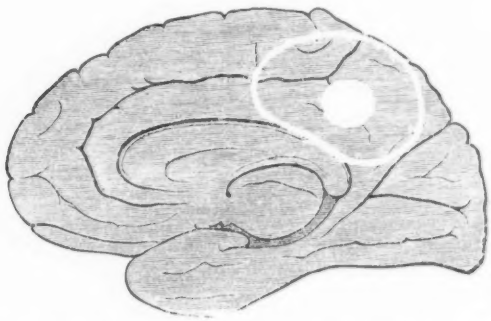
latter experiments, and it is in the belief that the following case adduces evidence of a similar nature that I am about to narrate it.

John W—, aged 47, a hawker, was admitted into the Paddington Infirmary on January 1st, 1892. He had drank a good deal of beer and spirits, but had had no previous illness until three years before admission, when he first began to have fits. He usually had fits in series of ten or more at a time; and, after one of these attacks nine months ago, the left hemiplegia, for which he was admitted, came on quite suddenly.

The patient on admission was quite rational, and when examined by my colleague, Dr. R. D. Hotchkis, was found to have partial loss of power and some rigidity in the left arm and leg, and it was noted that the sensation of the left arm was considerably diminished as compared with the right. Tactile sensation was very nearly but not absolutely lost in the left arm, normal in the left leg and elsewhere. The knee-jerk was normal on the right side, diminished on the left. No ankle clonus. There was weakness, but considerable power remained in the left arm and leg, and he was able to walk without assistance. The trunk seemed very weak, though we could not tell which side was worst, for he had difficulty in sitting up.

The left pupil was larger than the right, but there was no paralysis of cranial nerves. Five days after admission he had a fit. It was heralded, as usual, by a feeling of "cold water being poured on the left big toe, and running up the left side." First the left shoulder began to twitch, then the left leg was drawn up, and at the same time clonic spasms jerked the head and eyes to the right. This was immediately followed by general clonic spasms of the left arm and leg, the right arm and leg being meanwhile in a state of tonic rigidity. The eyes were wide open, conjunctivæ sensitive, teeth grinding, but the patient was completely unconscious. After lasting five minutes he recovered completely. He had another fit on the seventh, two on the eighth, one on the tenth, and one on the twelfth day after admission. He was extremely bad tempered, and became so unmanageable on the 16th January that he had to be transferred to the Workhouse. Three days later he became comatose, and after remaining in a condition of coma for seven days he died, without, so far as we were aware, any modification in his symptoms. The urine and viscera were noted as normal all the while. The temperature while in the Infirmary had a tendency to be subnormal. It was not taken in the Workhouse during the last week of life.

At the autopsy, made forty-eight hours after death, a tumour was found occupying the interior of the posterior segment of the brain, coming to the surface and involving the convolutions, as shown in the accompanying diagram. The other organs were natural, and there were no secondary deposits. The tumour was a round-celled sarcoma, about the size of a small orange, more or less spherical in outline, but its boundaries were not at all clearly defined anteriorly, infiltrating the adjacent tissues, especially at its upper and anterior boundaries. The light spot in the diagram



Median surface of right hemisphere of J. W—, aged 47. The light spot indicates the position where the tumour protruded from the surface. The line around indicates a zone of softening and congestion which projected above the level of the median surface.

indicates the position where the tumour protruded from the surface. The line around indicates a zone of softening and congestion which projected above the level of the parts around. This zone corresponds to the paracentral and quadrate lobules, and the posterior third of the gyrus fornicatus. The tumour also projected into the posterior and superior wall of the lateral ventricle. On the vertex of the brain the tumour did not appear, but the posterior third of the hemisphere was congested.

Remarks.—The diffuse nature of the anterior and upper parts of the tumour prevented one from saying very precisely which of the white fibres of the corona radiata were destroyed or involved; but it was quite certain that the grey matter of the posterior third of the gyrus fornicatus, the

greater part of the quadrate lobe, and the posterior extremity of the marginal convolution, was considerably disorganised and its communication with parts below cut off. Indeed the tumour fungated out on to the median aspect of the first-named convolution, which was the chief one involved. When read in connection with the experiments, and the case alluded to at the outset, one is justified in believing it *probable that the posterior part of the gyrus fornicatus is the cerebral centre for the tactile sensation of the arm.*

The paralysis from which the patient suffered was only very slight, and was quite in keeping with the supposition that the white motor strands were pushed aside, rather than destroyed, by the infiltrating growth.

As regards the "fits," which I had the opportunity of observing myself, it would have been somewhat difficult, from their character, to have localised the seat of the tumour. But this uncertainty is accounted for, as above mentioned, by the diffuse character of the anterior extremity of the tumour.

CASE OF HOMONYMOUS HEMIANOPSIA, WITH POST-MORTEM APPEARANCES.

BY EDWIN L. DUNN, B.A., M.B., ETC.

Assistant Medical Officer, Wakefield Asylum.

M. A. S., æt. 43, was admitted to the Wakefield Asylum on December 15th, 1891.

History.—Patient has been in the habit of giving way to bouts of alcoholic excess. For about a month before admission she had been drinking very heavily. About one week before admission she was noticed to be "paralyzed on the left side, the hand was drawn up and the leg useless." Nothing of her family history known.

On admission she was in a maniacal condition and raving wildly. No direct reply to queries could be obtained. It was noticed that there was paresis of the left side, with slight rigidity of elbow and shoulder joint. The grasp of left hand was very weak in comparison with the right. She could walk only with assistance, and the left leg was raised with difficulty and set down with a jerk on the heel. There was constant convulsive twitching of left leg and arm. The pupils were equal and semi-dilated. Their reactions were normal.

December 28th, 1891.—It is noticed that there is anæsthesia (to prick of a pin) of the left side, strongly marked in arm and leg, but not so much in face. There is possible slight ptosis of the left eyelid.

January 3rd, 1892.—Pupils are equal, all reactions present. The tongue is protruded to the left side. Sensation in left arm and leg is much better, being almost normal. Plantar reflex not well marked on left side; more marked on right, where it is, perhaps, excessive. Knee-jerk present on both sides, more marked on left. No clonus either side.

10th January, 1892.—Patient is much calmer. There is no defect of hearing. Sensation to pin-prick normal. Plantar reflex is excessive on both sides. There is no clonus. Knee-jerks normal. Rigidity of left arm almost disappeared. Grasp of left hand is weaker than right. There is no rigidity of left leg.

The gait is steady, but the left leg is moved in rather a jerky fashion, and there is a tendency to come down on the heel. There is no facial palsy. The tongue is protruded quite straight.

Eyes.—There is left homonymous hemianopsia, the defect being limited to the lateral half fields of vision in each eye. There is no change in the hemianopsic field with colours red or green. There is no colour blindness. Wernicke's pupillary inaction sign is not present (the pupil contracts when light is thrown on the blind half of the retina). Pupillary accommodative reflex present. Light reflex present, but sluggish. Concentrated light also sluggish. Consensuals present, but very sluggish in left eye. Pupils do not dilate to skin stimulus; they dilate at once emotionally. All ocular movements are perfect.

January 15th, 1892.—Hemianopsia unchanged. She says her leg is "all right," but complains that the left arm is stiff and the hand occasionally numb. She is fairly rational, but prone to emotionalism.

January 24th, 1892.—It is noticed that she is prone to drop pieces of bread, plates, &c., out of her left hand. She complains that it does not feel right. Grasp on both sides almost equal. She constantly leaves food which has dropped to the left side of her plate.

February 13th, 1892.—Mental condition and hemianopsia as before.

February 20th, 1892.—The left arm has never completely recovered its normal amount of motor power, but has always been slightly weaker than the right. Sensation to painful stimulus has remained unimpaired. She now complains that the arm is very numb, and is getting weaker. She has intense right-sided headache.

February 25th, 1892.—The headache has continued, the arm has become much weaker. Both pupils are widely dilated, light and consensual reflexes are present.

February 26th, 1892.—Patient became suddenly maniacal last night. To-day she cannot respond to queries, and is raving continually.

March 4th, 1892.—Mental state as above. The left arm and leg have been gradually getting worse, and are now completely paralyzed. Knee-jerk is present in both legs, is perhaps slightly increased in left. Plantar reflex is absent in left, present in right limb. There is some rigidity in paralyzed limbs, especially in the arm. Sensation cannot be tested owing to the mental state.

March 16th, 1892.—Sensation to prick of a pin is fairly per-

feet. She cannot tell what it is; says it is a "razor." She can just move left hand and can draw up the left foot one inch.

March 20th, 1892.—She had a slight stroke or syncopal fit yesterday. (Pallor, lividity, coldness of extremities, &c.) The left pupil is now widely dilated. Reacts slowly to concentrated light. The right pupil is semi-dilated, reacts quickly to concentrated light. Both appear to accommodate slightly and sluggishly. The optic discs and retinæ are found normal by ophthalmoscopic examination. The paralysis in arm and leg is incomplete as before. Sensation to painful stimulus appears perfect.

April 2nd, 1892.—Died of pneumonia, her condition not having otherwise changed.

Autopsy.—Cardiac valves and muscle healthy. Right lung congested with small spot of grey hepatization in middle of inferior lobe. Left lung solid and in a condition of grey hepatization. Liver and kidneys shew fatty change. Spleen soft and friable.

Skull cap thickened uniformly. Dura mater normal. No adhesions anywhere. Lepto-meninges thickened and softened, from right hemisphere peeled with undue ease, except from parietal and occipital lobes.

Right hemisphere of cerebrum.—The part of this hemisphere, from and including ascending parietal gyrus backwards, as compared with the left is notably swollen, softened, and collapsed. The pia cannot be stripped therefrom. Of this the superior parietal lobule, quadrate, first annectant gyrus, and lower portion of angular gyrus bounding the parallel sulcus, are the most disintegrated. The superior parietal lobule in front of the parieto-occipital fissure, shews a spot of ochre-coloured softening about the size of a threepenny-piece.

The supra-marginal gyrus consists of two parallel gyri taking origin from ascending parietal; the superior of these two is more softened than the inferior.

In the occipital lobe the superior occipital convolution is the most softened, and the mesial surface is not so softened as the outer. The ascending parietal is softened in its upper two-thirds only (*i.e.*, the part above the origin of the supra-marginal gyrus). The whole convolution is atrophied. The posterior third of the gyrus fornicatus is softened. The superior temporal gyrus is softened; its posterior two-thirds especially. The remaining temporal gyri and the frontal lobe appear healthy.

On horizontal section of brain the centrum ovale majus shews

softening in its posterior half. On further section the softening is found to involve the white matter of the parietal lobe and of the anterior two-thirds of the occipital lobe. The posterior third of the latter, though soft in comparison with the opposite side, is distinctly less affected than the parts first mentioned.

A section through the hemisphere passing through the three portions of the lenticular nucleus at the level of their greatest transverse diameters, shews that the greatest softening involved the posterior third of the roof of the descending horn, and the entire roof of the posterior horn of the lateral ventricle (*i.e.*, tapetum, optic radiations, and rectangular bundle of Wernicke). The white matter of posterior portion of occipital lobe is still of fair consistence.

On deeper section the tapetum, optic radiations, and rectangular bundle are still more disintegrated, readily breaking down under water; there is a small cyst the size of a pea in the posterior part of the roof of the descending horn near the margin.

Left hemisphere.—The convolutions are rather small, otherwise there is nothing abnormal.

No ruptured or thrombosed vessels discovered. Basal vessels healthy.

The cerebellum, pons, medulla and spinal cord shew no naked eye evidence of disorder.

Remarks.—It is possible that the primary lesion in this case was at the seat of the cyst in the posterior part of the roof of the lateral ventricle. We may regard this as the cause of the hemianopsia which was recognized early in the case and remained unchanged throughout. The cortex of the occipital lobe and the white matter immediately underlying the same—though undoubtedly affected by softening, was probably not so to such a degree as to cause well-marked symptoms. It is a noteworthy point that, although the angular gyrus was affected to an extreme degree, there was nevertheless no crossed amblyopia. Such a condition might possibly have been present on admission, but certainly was not so as soon as the patient's mental state permitted thorough examination.

With the condition of the left pupil, which was widely dilated for a fortnight before death, it is interesting to compare a case of Dr. Delépine's (read before the Pathological

Society of London, May 20th, 1890), in which there was right homonymous hemianopsia with dilatation of the right pupil—the *post-mortem* appearances being almost complete destruction of the left cuneus by softening, and also softenings in the middle part of inferior frontal and posterior orbital convolutions, and another softening at the bottom of the fissure of Rolando, opposite the posterior end of superior frontal sulcus.

I regret exceedingly that sensation was not more thoroughly tested. The patient's mental state, however, and her extreme emotionalism rendered any lengthened examination requiring her attention extremely difficult. Sensation to the prick of a pin was normally present throughout except for the first few days. This fact, as far as it goes, is opposed to the views of Munk, Luciani and others, who hold that the localization area for common sensations lies in the central region of the cortex just beneath the parietal bone, a region in this patient extensively diseased. It is interesting, however, in connection with the experiments of Horsley and Schäfer (*Trans. Royal Soc.*, vol. 179 (1888), B., pp. 1-45) who hold that the centre for painful and tactile sensations is in the gyrus fornicatus. Though part of that convolution was in this case diseased, there is no reason to suppose that sufficient healthy tissue was not present to functionate correctly.

As to the awkwardness and subjective sensations of the left hand, this is an interesting point, and was a prominent feature of the case throughout that period in which the patient was fairly rational. She constantly complained to the nurse that she had not proper use of her left hand, that it was numb and not like the other—this being a source of great distress to her. Her awkwardness too in holding her sewing, and her tendency to let objects drop was remarkable, although at this time the grasp of the hand was fairly good and there was little diminution of the motor power of the arm. In this connection it is interesting to note that Bernhardt (*Archiv. fur Psychiatrie*, vol. xii., p. 780) reports four cases in which there were subjective affections of sensibility

Fig. 1.

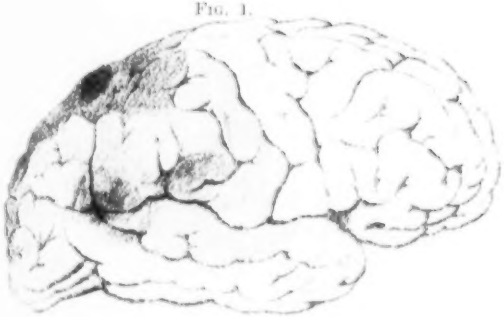


Fig. 2.

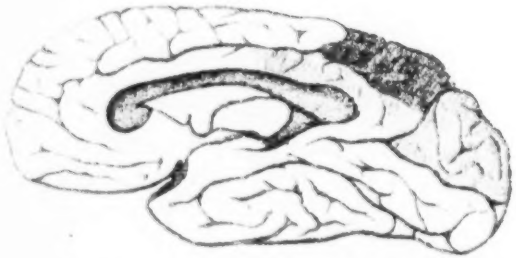


Fig. 1.—Exterior surface of R. hemisphere.
Fig. 2.—Mesial surface of R. hemisphere.

Fig. 3.

Fig. 3.—Deep horizontal section of R. hemisphere, shewing softening in posterior part of roof of Lat. Vent.

Depth of shading indicates degree of softening.

and impairment of the muscular sense associated with hemianopsia, and compares these with a case of Samelsohn's, shewing similar disturbances. He considers that these cases belong to a type in which after an apoplectic attack the symptoms of paralysis disappear, leaving a characteristic awkwardness in the limbs of one side, especially the upper extremity. This awkwardness is further aggravated by the hemianopsia, a frequent symptom in these cases. These cases of Bernhardt's have not as yet been completed by *post-mortem* examination. It is worth while also to compare this case with one of Westphal's (*Charité Annalen*, 1882), in which there was marked impairment of the muscular sense of the right upper extremity, associated with diminished right-sided sensibility and bilateral right hemianopsia. The lesions found after death were softening and atrophy of the whole parietal lobe (including ascending parietal), the greater portion of the occipital and a small portion of the temporal lobe, the cortex only being involved.

The phenomena exhibited by the left plantar reflex (which was at first deficient, then present, and disappeared totally after the second maniacal attack, about one month before death), may be of interest in connection with the disease of the white and grey matter of the opposite hemisphere and the macroscopically healthy basal ganglia.[1]

[1] Königsdorf (*Deutsche Med. Wochenschrift*, March 3rd, 1892) reports a case of hæmorrhagic encephalitis in which, with much softening and blood extravasation into the grey matter and white substance of the left hemisphere, the basal ganglia also being affected, there was three days before death complete absence of all superficial reflexes on the right side.

A CASE OF CEREBELLAR TUMOUR, WITH LOSS OF THE KNEE-JERKS.

BY HENRY HANDFORD, M.D., M.R.C.P.LOND.

Physician to the General Hospital, Nottingham.

CASES of brain tumor, are always interesting, on account of the wonderful variety of symptoms to which they give rise, dependent not only on the position of the tumour but also very largely on its degree of vascularity, its rapidity or slowness of growth, and on pressure, sometimes affecting parts at a considerable distance, sometimes in an inexplicable manner, doing but little damage to nerves or tissues in close proximity to or involved in the growth. And, further, the symptoms are apt to be complicated by secondary meningitis which so frequently arises, and by the accumulation of fluid in the ventricles, or in the sub-arachnoid spaces. But however interesting the individual problems may be, it is not on that account that I bring forward the following case, but rather as a contribution to the elucidation of the vexed question of the physiology of the knee-jerks.

Case.—L. W., aged 16, was admitted on March 2nd, 1891. His illness began three months previously, with sickness coming on many times daily, independently of taking food. This was followed by headache, chiefly frontal, giddiness, unsteadiness of gait, and occasional tingling in the hands and feet. For the ten days before admission he suffered from fits, commencing with pain in the back and head. He then suddenly "shoots himself out straight" and lies thus quite still with his eyes wide open for ten to twenty minutes. Beyond very slight twitchings of the hands no spasmodic movements or muscular contractions take place during the fits, the limbs remaining flaccid. For the last few months his sight has been failing, and for the past four or five days he has seemed "strange in his mind." He is very slow in answering a simple question. On admission it was noted that the knee-jerks were completely absent on both sides. They

were thoroughly tested, as I was much surprised at the fact, and could not then offer a satisfactory explanation. The plantar and abdominal reflexes were present on both sides, and there was very slight ankle clonus also on both sides. There was no anæsthesia or analgesia. The other systems were normal, and the urine was free from albumen and sugar. Double optic neuritis was found, more advanced in the right eye, and by March 9th some weakness of the ocular muscles, especially of the abductors, and slight nystagmus were noted. The pupils were dilated and reacted imperfectly to light. There were numerous small hæmorrhages in the retinæ. He could still see a little, but eventually he became quite blind. He was rather feeble, but there was at this time no definite local paralysis. His gait shewed much inco-ordination and unsteadiness with a tendency to stamp the heels. He had great difficulty in turning round, and complained much of giddiness. He could stand with difficulty with the feet apart and the eyes closed, but with the feet close together he tended constantly to fall *backwards*. He had never fallen whilst walking with the eyes open. On March 13th there was marked internal strabismus of the right eye, but it was temporary.

April 4th.—The patient gets rapidly worse. He screams a great deal, and lies in a semi-conscious state. There is a good deal of retraction of the head. He has had several more fits. Knee-jerks still quite absent, though the plantar and abdominal reflexes are still brisk. No ankle clonus can now be detected on either side. On account of the imperfect consciousness it is very difficult now to determine the condition of sensation. He cannot move body or limbs. He is emaciating rapidly. The temperature has all along been normal.

After this the paralysis became more and more absolute, difficulty in swallowing arose, and also attacks of dyspnœa, and hurried respiration, apparently from implication of the respiratory centre. Although emaciation was extreme, and he had been accustomed to pass urine and fæces into the bed for several weeks, no bed sores formed. The limbs remained flaccid. He died on May 31st. Total duration of symptoms six months.

Unfortunately the *post-mortem* examination took place when I was out of town for a day or two, and so the spinal cord was not examined.

There was no meningitis, the cerebral convolutions were somewhat flattened, the lateral ventricles distended with fluid, and a tumour was found about the size of a large walnut on the under

surface of the cerebellum growing from the middle lobe (fig. 1). It was pressing on the medulla, which was somewhat flattened, and it had closed the foramen of Majendie, thus causing the distension of the lateral ventricles. It did not involve any of the cranial nerves (fig. 2). On antero-posterior vertical section the tumour was found to be more or less defined, though not encapsuled. It had destroyed the whole of the middle lobe of the cerebellum and extended into both lateral lobes, especially the left. On microscopic examination it proved to be a sarcoma. There were no other growths in any of the other viscera.

The physiology of the knee-jerks is still obscure. It is agreed that the integrity of the reflex arc, consisting of afferent nerves, ganglion cells in the anterior cornua, and efferent nerves is necessary, though it is denied that the movement is a true reflex action, because the time elapsing between the stimulus—the blow on the tendon—and the jerk of the leg is much too short. But it is not agreed whether anything further is necessary for the normal activity of the knee-jerks, nor why they become excessive in some cases and fail in others, the reflex arc remaining uninjured.

According to one school the spinal reflex arc is sufficient by itself for the production of the knee-jerks, and it is habitually controlled by higher encephalic centres. When this controlling influence is cut off by a cerebral lesion, or by a total transverse lesion of the cord, exaggerated reflexes result.

On the contrary, the French school teach (and their views have been largely adopted in this country), that the degenerative changes in the terminal portion of the fibres of the crossed pyramidal tracts "cause an irritative overaction in the related great ganglion-cells in the anterior cornua, and thus lead to an exaggerated condition of 'tonus' in the muscles and the increased tendon reactions."

Dr. Hughlings Jackson has taught for many years that muscular "tone" and the tendon reactions depend on cerebellar influence, which is normally more or less antagonised by the influence of the cerebrum. And when the influence of the cerebrum is cut off by any lesion "the unantagonised influx of cerebellar energy" is the cause of the increased "tonus," tendon reactions, and rigidities.

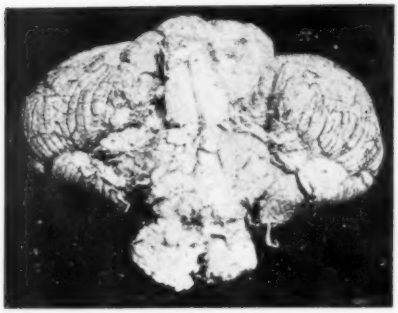


FIG. 1.

Photograph of the under surface of the Medulla, Pons, and Crura lying upon the Cerebellum, the middle lobe of which is occupied by a tumour projecting chiefly to the right. *(Hold at two feet distance.)*

FIG. 2.

Vertical antero-posterior mesial section shewing the extent of the tumour and the flattening of the Medulla. The tumour is dark.

have been any peripheral neuritis, sclerosis of the posterior columns of the cord, degeneration of the posterior nerve roots, or any tumour or other coarse lesion of the cord or cauda equina to account for the loss of the knee-jerks. And, besides, with the exception of this one disputed question of the knee-jerks, the cerebellar tumour and the consequent distension of the lateral ventricles, were quite sufficient to account for all the symptoms from which he suffered. There seems, therefore, much reason to believe that the loss of the knee-jerks in this instance was due to the cutting off of the cerebellar influence, by the growth of the tumour.

LARGE TUMOUR OF THE PITUITARY BODY, INCREASED KNEE JERKS, NO ACROMEGALY, NO GLYCOSURIA.

BY HENRY HANDFORD, M.D., M.R.C.P.LOND.

Physician to the General Hospital, Nottingham.

Case.—E. S., aged 53, single, was admitted in July, 1891. She began to lose the sight of the right eye last Christmas. The left failed at Easter, since which time she has been unable to follow her employment as a lace worker. Vomiting has been frequent, and she has suffered greatly from headache, chiefly affecting the right parietal region, which is tender to the touch. She lies in a drowsy state with the eyes closed. Memory is very defective, but otherwise her intelligence is acute, and it remained so until the end. There was no trace of acromegaly; and the urine remained free from sugar as well as from albumin. The eyelids could be raised but not quite fully, and there appeared to be some weakness, though the patient professed that she kept them closed voluntarily. There was also slight weakness of the internal recti, and some nystagmus on looking towards the right, none on looking towards the left. The pupils were equal, one half dilated and insensitive to light. The patient was blind but could distinguish day from night, though she could not distinguish the position of the windows. There was optic atrophy, with white discs, rather small vessels, but no trace of previous neuritis. At this time there was some doubtful weakness of the left side of the face, which a few weeks later became marked. There was no definite weakness of the limbs, the patient being able to walk fairly and use both hands. There was no alteration of sensation. The plantar reflexes were not noticeably increased, but the knee jerks and arm jerks were exaggerated. The bowels were exceedingly obstinate, being only moved after croton oil. She had no fits, but frequently had attacks of stertorous breathing which lasted several hours.

On August 31st, it was noted that the tongue deviated towards the right, and that the right half appeared wasted. She was much more feeble and could hardly stand. By September 21st the left arm and leg appeared decidedly weaker than the right, but on October 1st this difference could not be definitely estab-

lished, so much did the degree of paralysis vary. Taste was impaired and she called salt sour; but other things she recognised. Hearing remained acute. Speech became more and more thick and eventually she lapsed into a comatose state and died on October 17th, three and a half months after admission, and ten months after the first onset of symptoms.

At the necropsy a large tumour was found at the base of the brain pressing on and causing considerable flattening of the Pons Varolii, especially in its superior half. On antero-posterior vertical section the tumour was found to have originated in the pituitary body, and to be more or less encapsuled. The arteries of the circle of Willis were imbedded in the middle of the growth. The optic nerves and commissure were pressed very flat, and were partially involved in the tumour. The 3rd and 4th nerves were flattened and wasted, and the crura cerebri were also flattened and much atrophied. The tumour was a sarcoma. There were no growths in any other parts of the body.

The only mental symptoms here were some oddness of manner, great loss of memory and hebetude. It is remarkable that so large a tumour in that position should have caused so little injury to the cranial nerves. The optic atrophy was probably due to the pressure on and wasting of the optic nerves and commissure, and was not the result of previous papillitis, because the vessels were very little diminished in size, and there was no trace of lymph remaining. With this exception none of the other cranial nerves were *completely* paralysed. So far as the rest were affected the paralysis was *one sided*, or *variable* and incomplete, although the tumour was large, centrally placed, and nearly symmetrical. Although the crura cerebri were so much flattened there was little paralysis of the limbs until the last week or two, and no rigidity. The knee jerks and the reflexes of the upper extremities were increased on both sides as would be expected. It is to be noted that while the cerebral influences were largely cut off by the pressure on the crura the cerebellum and its connections with the cord were not affected.

The associations of tumours of the pituitary body or hypophysis cerebri, both with acromegaly and with glycosuria make the absence of both these conditions in this case worthy of note.

FIG. 1.

Photograph of the base of the brain. The whole of the middle fossa is occupied by a mushroom-shaped tumour. *(Best seen at two feet distance.)*

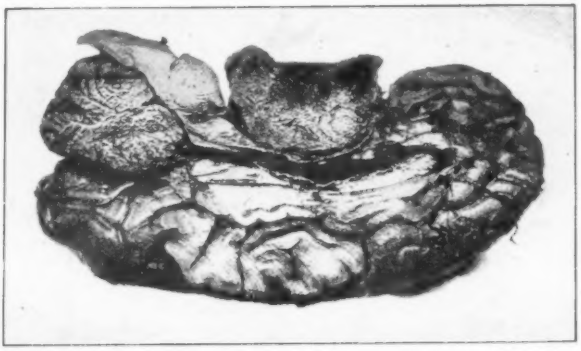


FIG. 2.

Vertical antero-posterior mesial section shewing the flattening of the Pons Varolii and of the Crura Cerebri by the pressure of the tumour which is growing from the Pituitary Body.

TUMOUR OF PITUITARY BODY WITHOUT ACROMEGALE.

BY ERNEST WILLS, M.D.LOND.

Assistant Medical Officer, County Asylum, Rainhill.

J. G., æt. 26, cotton operative, was admitted into Rainhill Asylum, April 20th, 1891, suffering from dementia; he was very confused, his memory was defective, rendering him incapable of giving any good account of himself, but he complained much of pains in his head. His facial expression was dull, somewhat anxious, and his general appearance cachectic. He looked younger than his age, was 4ft. 10in. in height and weighed 100lbs. His brother, who accompanied him to the asylum, also a short man, but taller than patient, healthy and fairly intelligent, gave the following information:

Family History.—They were both illegitimate children, mother dead, cause unknown, father living, healthy, now married. No insanity, consumption or cancer in family on either side as far as informant knew.

Previous History.—Patient had never had any severe illnesses, or chest trouble. He had complained of pains in his head since he first began work at the mills fifteen years before. During the last six months they had become much worse, causing him to cry out at times "Oh, my head," and he had become very stupid. There had been no previous attack of insanity and no fits.

Condition on Admission.—Temperature normal. Well nourished generally. Skin elastic, subcutaneous fat abundant. Circulatory system, heart sounds normal, pulse regular, moderate tension 68. Respiratory system normal. Abdomen normal. No signs of syphilis. No albumen or sugar in the urine. Nervous system. External muscles of right eye normal, some paresis of left internal rectus with external strabismus. Irides hazel, margins circular and regular. Right pupil reacts to light and accommodation,

left scarcely to either. Very little if any vision with the left eye. No paralysis of facial muscles, tongue protruded in middle line. No marked paralysis of limbs, but the grasp is distinctly feeble on both sides and a little weaker on the left than right. He is feeble on his legs, walking with a staggering gait and slight tendency to fall backward. Reflexes: the knee-jerks are present on both sides, the left being somewhat excessive. No ankle clonus. Superficial reflexes present, average, no marked difference between the two sides. Sensation to touch pain, and temperature is good all over. No hyperæsthesia. There is consecutive optic atrophy of considerable duration in both discs, but especially well marked in the left.

April 30th.—Very dull and heavy, lies in bed taking no notice of surroundings, crying out with pain occasionally. Has vomited two or three times. Has had no fits.

May 5th.—Brighter and more lively; although still confused and complaining of headaches, says the pains are less severe.

May 12th.—Complains of recurrence of severe pains in the head and also in region of the left elbow; there is no neuritis. Vomited once to-day. No fits.

May 16th.—He was very dull and lethargic, and had retraction of the head. After some hours he became comatose and died at night somewhat suddenly.

The following account of the tumour is from the report of the autopsy Dr. Gordon Sanders' (late pathologist to the asylum).

Post-mortem Examination.—Body squarely built, the hands short, broad and somewhat spade like, but all limbs fairly proportioned. There was a very large subcutaneous deposit of fat. There was nothing of any importance in any of the organs except the brain. The scalp was thick and hairy, the skull low, broad, rounded, of good symmetry, and natural thickness. On opening the skull and dura mater the cerebral hemispheres were seen to be flatter than natural; on attempting to remove the brain a tumour was found at its base, in the region of the infundibulum, consisting of a dark soft greenish-yellow mass. The tumour could not be removed entire as a large part of it was wedged in the sella turcica, it was therefore cut across and this part dissected out afterwards. The brain itself seemed fairly normal, and presented no irregularity of convolutions, although on the vertex they seemed somewhat swollen and flat with considerable distension of veins; there was distinct diffused opacity of membranes, however, with much adhesion, in many parts reaching actual decortication, especially at the vertex. At the base of the brain within the

interpeduncular space lay the tumour, extending anteriorly as far as the posterior termination of the orbital surface of the frontal lobes. Laterally it was overhung by the temporo-sphenoidal lobes. Posteriorly it extended to and separated widely the crura cerebri. The greater mass of its substance lay eccentrically to the left and on separating the (apparent under but real) upper surface was found to lie in a hollow between the left frontal lobe and the left crus cerebri.

Position of Structures Round.—Anteriorly the two *olfactory nerves* were thin and flattened, the left much the more so, of which the external root was alone visible as a thin whitish band. The right *optic tract* skirted the right side of the tumour and was involved in the thickening of the capsule, the half of the optic chiasma on that side was seen lying just anterior to the projecting part of the tumour; it was white, tough and atrophied. The *left optic tract* was very remarkably altered. It passed over the crus as a pale yellowish thin band and at once became involved in the tumour mass, being distinguished as a soft yellowish ill-defined band passing forward obliquely to join the right tract in the chiasma, the left half of the chiasma being soft, yellowish-grey, and translucent in marked contrast to the right. The *crura cerebri* were widely separated, the right was flattened and wasted, the left more displaced outwardly, more rounded and prominent. The *corpora albicantia* were pushed to the right side and much flattened, the right being nearly half the size of the left. The floor of the third ventricle and interpeduncular space seemed softened by pressure, but were not implicated in the tumour. The *pons* seemed wasted and narrowed, the ventral pyramids were prominent, but the mesial sulcus was deep and the sides flattened. The *medulla* was possibly somewhat atrophied.

Description of Tumour.—It consisted of two parts connected by a pedicle, the upper part was removed with the brain, the remainder lay in the sella turcica and was dissected out after, and the sphenoid bone removed. The portion of tumour removed with the brain consisted of a soft glistening body the size of a Tangerene orange irregularly rounded or ovoid, about 3.5 cm. in diameter and was divided into two unequal parts; a soft flatter darker basal portion of diameter referred to, and a more prominent hard yellowish spongiform body the size of a marble, somewhat gritty, seated in the right anterior segment (apparent left, as the brain lay with tumour uppermost), separated by narrow neck from the basal portion. This second projecting portion was that which was continuous with the pituitary body and infundibulum. The

whole tumour had a thin distinct capsule unequal in thickness, more ill-defined and grayish over the basal part, but tough, thicker and whitish over the dependent portion, from which it was easily separable. The flatter basal portion was of varied colour; the left half being of greenish yellow hue, somewhat like a greengage in colour, across it posteriorly lay a large whitish yellow band, which proved to be the left optic tract; the right half was more distinct, yellower and firmer.

The bony floor of the sella turcica presented sharp elevations and ridges, and when the dura mater was stripped from them their summits were found rough and granular. The sella turcica was much broadened and its constituent bones remarkably thin, the basilar portion being a mere lamina and very brittle. The tumour here was very soft in its anterior half and lay entirely enclosed in a thick capsule, the posterior half was of deep canary yellow, rough, spongioid and the seat of a fairly recent hæmorrhage. The anterior half or section was found to be a cyst containing a greenish yellow glistening fluid somewhat viscous. Although the floor of the sella turcica was pitted in several places into cavities the size of a split pea, at no point was there any communication with the buccal cavity from which the tumour was shut off by a mere shell of bone.

Microscopic examination of various parts of the tumour showed that it was composed of two parts, a minor solid and a much larger part, in fact the majority of the tumour, which was cystic. A section of the solid or semi-solid portion of the tumour exhibited a structure resembling that of the thyroid body, viz., a capsule of fibrous tissue with cleft like spaces in it lined with flattened epithelium, an inner stroma of fibrous tissue containing small cysts of various sizes lined with columnar epithelium and the lumen of the cyst containing a viscid substance or filled with cells columnar, ovoid, or round. Nowhere in the specimens were any columns of cells spreading in a malignant manner, although there were here and there to be seen groups of cells somewhat resembling the nests in a carcinoma, these were probably only the walls of smaller cysts in the plane of the section.

Sections of the walls of the larger cysts only showed a fibrous structure, with the cleft like spaces as mentioned above, and with patches of pigment here and there, the remains probably of old hæmorrhages as the fibrous tissue was very vascular.

Commentary.—This case has several points of great interest which make it worthy of record. 1. Clinically from the large size it attained, and the amount of displacement of surrounding parts it caused without giving rise to any serious symptoms until quite late in the case. 2. The nature of the tumour itself. 3. The absence of any acromegale in association with the tumour of pituitary body.

Clinically the principal symptoms were those usually found with cerebral tumour, headache, vomiting and optic neuritis with consecutive atrophy; but until his mental condition caused him to leave off work, he had managed very well as an operative in a mill and at no time had he any paralysis, nor did the almost total loss of sight with the left eye seem to have troubled him.

The organic dementia caused by the tumour is interesting, too, as most authorities agree that cerebral tumour as a cause of insanity is decidedly rare, although insanity as a sequence of tumour is fairly common. With respect to this point it may be mentioned that during the last twelve months there have been an exceptional number at Rainhill Asylum, viz., five cases in about 240 *post-mortems*—this of course is very much above the average.

With regard to the tumour itself, it seems to be what is called by some authors, goitre of the pituitary body from its resemblance to the structure of the thyroid gland. Weigert calls it adenoma of the pituitary body and supposes it to be a cystic degeneration and hyperplastic overgrowth of the anterior lobe, which lobe in the adult normally contains a large number of convoluted tubules or alveoli lined with columnar epitheluim which in some cases fills up the tube; moreover portions of the tubules are frequently cut off by the connective tissue so as to form vesicles. The lymphatics of the organ originate in cleft like spaces between the tubules and pass to a network in the capsule (Quain's Anatomy, vol. 2). All these characters are represented in the present tumour. With regard to acromegale, in many cases of that disease which have been published, hypertrophy of the pituitary body has been observed, and Dr. Pierre

Marie, in his paper on the subject, BRAIN, July, 1889, says, "Finally amongst the lesions affecting other organs, and which, after what has been observed in other autopsies, seem to me to be constant in acromegale, must be mentioned hypertrophy of the pituitary body with enormous dilatation of the sella turcica, persistence of the thymus and finally hypertrophy of the cord and ganglia of the sympathetic system." The two latter points were not specially observed, but there were certainly no signs of acromegale about the patient, therefore as a negative case it seemed worth recording.

THE STUDY OF LARYNGEAL PARALYSIS SINCE THE INTRODUCTION OF THE LARYNGOSCOPE.

BY FELIX SEMON, M.D., F.R.C.P.

THE following remarks contain the substance of an article which I contributed to the "Festschrift," dedicated to Professor Virchow on the occasion of his 70th birthday. As this paper in all probability will be accessible to comparatively few members of the profession in this country, a translation of its contents may not be unwelcome to those neurologists who have no time to study the history of this subject in detail. This English edition is on the whole a faithful reproduction of the German original; the introduction, however, which referred more specially to the particular occasion of its origin, has been omitted; and the papers of Wagner, Burger, Krause and Risien Russell, recently published in full, are considered at greater length.

I do not aim here at a complete bibliographical survey of the subject. The period I cover extends from the introduction of the laryngoscope, thirty years ago, down to the end of 1890, in addition to which the four important papers just mentioned and published in the course of 1890 and 1891 are discussed, and my object is rather: to retrace in broad lines the evolution of our knowledge concerning laryngeal paralyses, and to refer in detail to such contributions only which in my opinion have actually contributed towards this development.

Opinions will, of course, differ as to the relative value of the individual contributions and attribute to some a greater or a lesser importance than has been accorded to them in the following lines.

But, as I have contended already on a previous occasion (1), these are points on which everybody has the right to form an opinion of his own; and I can give the assurance that I have followed the best of my judgment, being inspired only by the desire to do justice to everyone concerned.

With the only exception of the subject of benign neoplasms, probably no part of laryngeal pathology has profited so much by the introduction of the laryngoscope, as that dealing with the

motor neuroses of this organ. The course of the other most prominent laryngeal affections, such as tuberculosis, syphilis, malignant neoplasms, &c., always offered points for the diagnosis of the true nature of the local disease. But the main, or rather the only, phenomena by which motor laryngeal paralyses are usually manifested, viz., disturbances of phonation, aphonia, dyspnœa, are of such multifarious significance as to point to an inflammatory lesion or to a new growth, as much as to a paralysis. In prelaryngoscopic times one had therefore to depend entirely upon mere suppositions concerning the nature of the laryngeal affection met with in such cases, and von Ziemssen is perfectly justified in stating (2) in the introduction to the chapter on Laryngeal Neuroses of his "Cyclopædia," that the older literature on this subject is devoid of any importance.

The distinction of having diagnosed the first laryngeal paralysis by means of the laryngoscope belongs, so far as I know, to my illustrious teacher, L. Traube.

In Göschen's "Deutsche Klinik" (3) he described the laryngoscopic appearances in two cases of aneurism of the aorta, viz., paralysis of the left vocal cord, and in his commentary explicitly stated that these laryngoscopic appearances had at once drawn his attention to the possible presence of an aneurism. Thus already in the first two cases of laryngeal paralysis, ascertained by means of the laryngoscope, the *diagnostic* value of the method was emphasised.

Within the next few years the number of communications referring to motor laryngeal disturbances quickly increased. In 1863, Gerhardt's classic "Studies and Observations of Paralysis of the Vocal Cords" (4) were published, which contain numerous valuable cases and in which the principle already is stated, that in cases of paralysis of the trunks of the vagus or recurrent laryngeal nerves, individual strands of fibres may be affected singly or at least predominantly, a statement which was opposed very energetically six years later by Navratil, (5) and as late as seventeen years afterwards by Bosworth. (6)

During the years 1863 to 1876, a large series of valuable communications were published, which increased our knowledge concerning the individual forms of laryngeal paralysis, without however, formulating any general principles concerning the laws governing the production of these different forms. Apart from Gerhardt, who has paid special attention to this chapter up to the present (7), Schnitzler (8), Bäumler (9), George Johnson (10), Von Ziemssen (11), Morell Mackenzie & Evans (12), Riegel (13),

Oliver (14), and Nicolas Duranty (15) must here be mentioned. The contributions of Bäumler and George Johnson are specially important from the standpoint of principle, inasmuch as these two authors showed even at that early period of more recent investigation, that a *uni*lateral affection of the *vagus* may under certain circumstances produce a *bi*lateral paralysis of the vocal cords, or paralysis on the one side, and spasm on the other. Their observations have subsequently been corroborated by McCall Anderson (16), Whipham (17), and Kurz (18), whilst the cases described by Schnitzler (19) and Sommerbrodt (20), in which, according to these authors, a *uni*lateral paralysis of the *recurrent* laryngeal had produced a *bi*lateral paralysis of the vocal cords, were erroneously interpreted, as I have shown (21) in connection with Sommerbrodt's case.

In the period between 1860 and 1870 a number of special text-books were published, in all of which the paralyses of the larynx are dealt with, in more or less detail.

The first place probably belongs to the work of Türck (22), which in addition to many well-illustrated and accurately observed cases, contains an attempt at an etiological classification and precise subdivision of the several forms of paralysis.

In 1866 there appeared a little work by Morell Mackenzie (23) on motor disturbances of the larynx. It is distinguished by its casuistic wealth, and deals more specially with the method of direct electrisation of the vocal cords by means of the author's laryngeal electrode, but suffers from the mistake that organic paralysis of the trunks of the laryngeal nerves is throughout confounded with functional paralysis of the adductors of the vocal cords. The same error deprives the industrious chapter on paralyses of the larynx in J. Solis Cohen's (24) great text book of much of its value.

To the same period belong the experimental contributions of Philip Schech (25) and G. Schmidt (26); the former will always remain a laryngological classic, inasmuch as it contains, in addition to its bibliographical treatment, numerous carefully conducted and critically discussed experiments on the functions of the individual nerves and muscles of the larynx, and on the bearing of these physiological data upon the pathology of laryngeal paralysis. We may note that Schech already thinks of the existence of a reflex-tonus of the posterior crico-arytænoid muscle, when discussing the position of the vocal cords during quiet respiration—a point inexplicable at the time, and to which we shall presently return.

From about 1876 the study of laryngeal paralyses was directed to an interesting form, characterised by loud sonorous inspiration, severe and purely inspiratory dyspnœa, perfectly preserved voice and free expiration. I speak of that affection, which up to Krause's paper, which will be fully discussed hereafter, was generally spoken of as "bilateral paralysis of the abductors of the vocal cords." It is true that already, at an earlier period, such cases had been reported; the first by Gerhardt in 1863 (*loc. cit.*, pp. 268 and 269); the next—a doubtful one—by Türck (*loc. cit.*, p. 461); the third by Morell Mackenzie and Hughlings Jackson (27); the fourth also by Mackenzie (28); the fifth by Riegel (29); yet it was only the classical paper on "Respiratory Paralyses" (30), contributed by the last named author which in 1876 attracted the general attention of observers.

But even in this paper, as well as in other contributions to the question (Penzoldt (31), von Ziemssen (32), Burow (33), and Bosworth (34), no attempt was made to trace to general laws the curious fact that in affections, which apparently concern the *entire* centres or trunks of the motor nerves of the larynx, only the *abductors* of the vocal cords or the nerve twigs which supply these muscles are found to suffer. On the contrary all these writers only sought to explain by some hypothesis or other the apparent pathological curiosity, apart from any other correlated instances.

Only at a later period was it found that the phenomenon is but an example of a definite law, and this fact was discovered in the following manner :—

In the years 1876 to 1878 I had the opportunity of observing at the Hospital for Diseases of the Throat a series of six cases of bilateral paralysis of the glottis-openers which I described in April, 1878, in a paper (35), read before the Clinical Society of London. In some of these cases the cause of the disease could not be traced definitely; in others, however, the clinical or *post-mortem* investigation proved that central or peripheral affections had been present, which evidently had involved the *entire* centres or trunks of the motor laryngeal nerves. Nevertheless in these cases the appearances of isolated paralysis only of the glottis-*openers* had been present during life; after death the posterior crico-arytænoid muscles only, or at any rate to a much higher degree than the glottis-*closers*, were found to have undergone fatty degeneration and atrophy, whilst in the nerves some normal nerve fibres were found among numerous degenerated ones.

Whilst I was studying these cases of *bi*lateral paralysis of

the glottis-openers an even larger number of cases came under my observation, in which the same phenomenon was observed *uni*laterally, *i.e.*, in which isolated paralysis of one posterior crico-arytænoid muscle was seen combined with lesions of the entire nerve trunk or its medullary centre.

The *constancy* of this phenomenon necessarily attracted my attention, for whilst the exclusive appearance of paralysis of the *abductors* was so frequently seen in organic lesions of the centres or of the nerve trunks, I did not once see the reverse occur under similar circumstances, *i.e.*, isolated paralysis of the *adductors* of the vocal cords.

At that time we all were entirely under the influence of the doctrine which had gradually become developed in the course of the preceding 18 years, that, whilst in central or peripheral lesions of the entire motor apparatus of the larynx individual muscles *could* be *exclusively* affected, yet it depended more or less upon *accidental* circumstances of the individual case *which* of these muscles would be paralysed. This doctrine was advocated as late as 1876, in Von Ziemssen's Cyclopædia (36). My own experiences, however, as shown above, seemed to contradict it so decidedly that I instituted a thorough search through laryngological literature, in order to find out whether these experiences were *exceptional*, or whether indeed the abductors of the vocal cords and the nerve twigs leading to them were really prone to much earlier and more frequent affection than the adductors, in progressive organic lesions of the entire centres and trunks of the motor laryngeal nerves.

The result of this search greatly surprised me. For according to the current doctrine the number of cases in which an initial incomplete paralysis of the recurrent laryngeal nerve manifested itself by the symptoms of a paralysis of the *ab*ductors, ought to have been equal, or nearly equal to that of the cases in which the adductors were chiefly affected. Now, it resulted from my enquiry, that *not a single* really valid case, at any rate not one authenticated by *post-mortem* examination, could be found in the whole domain of laryngological literature, in which organic progressive lesions of the nuclei or trunks of the motor nerves of the larynx had manifested itself by an initial paralysis of the *ad*ductors. On the other hand I found quite a large series of recorded cases, in which under similar conditions in the commencement of the disease and often up to the death of the patient, the symptoms of paralysis of the *ab*ductors had manifested themselves and in which after death atrophy and degenera-

tion of the posterior crico-arytænoid muscles only (or at any rate more marked than in the adductors) had been present. In other words, my own observations were not, as I had originally believed, *exceptional*, but conformed themselves to the *generalised* sum of previous experiences.

Even before I had arrived at this final result, an opportunity offered itself of publishing the experience I had so far gained. As I have already stated on a previous occasion (37), in Sir Morell Mackenzie's work, which was just then about to be published, and of which I edited a simultaneous German edition, the author had adopted von Ziemssen's view, that it depended more or less upon *accidental* circumstances of the individual case as to *which* set of fibres of the motor laryngeal nerves would first be paralysed. It was only after I had communicated the results I had so far obtained to Sir Morell, that the paragraphs dealing with the subject were altered by him in the sense of acknowledging and emphasizing the greater frequency of initial paralysis of the *abductors* (38).

The footnote (p. 574) which I added to his statement in the German edition incontestably proves that I had already at that time clearly recognised the *importance* and *bearings* of the whole question.

This footnote runs as follows:—

"From the above it will be seen that the author assumes a differentiation to exist in the centre itself of the nerve twigs supplying the individual muscles of the larynx. The German editor agrees with this view on theoretical grounds, but wishes to draw attention already here to the fact that even such a hypothesis does not suffice to explain a most remarkable phenomenon, viz., that in many cases in which *central* or *peripheral* lesions unquestionably affect the whole nerve (most frequently in cases of pressure upon the entire trunk of the recurrent laryngeal in cases of aneurism of the aorta) the symptoms of paralysis of the glottis-openers only are seen during life. Whilst this has been observed by the author as well as by the editor in a considerable series of cases (several of them will be fully related further on), *the latter knows of no case* in which, after paralysis during life of one or several of the adductors *post mortem examination has shown disease of the entire nerve trunk*. This proclivity of the fibres going to the abductors to be affected exclusively or long before the others, is very strange."

I hope I need make no excuse for recapitulating here the stages in the development of this topic. I have been so frequently

asked how I had arrived at formulating the law that I must conclude that my previous descriptions of its origin have not been exhaustive enough; moreover, this historical sketch will enable every unbiassed reader to form his own opinion on the unpleasant and long continued discussions between Professor O. Rosenbach and myself as to the question of priority.

Before the publication of Mackenzie's work and of my own first special paper on this point, Rosenbach had observed a case of bilateral paralysis of the vocal cords due to carcinoma of the œsophagus, originally presenting a paralysis of the glottis-openers, which gradually passed over into total paralysis of the recurrent laryngeal nerves, and on the strength of this single observation had formulated this conclusion (39): "Above all, the fact must be registered that *in compression of the trunk of the recurrent, the function of the abductors suffers first*, and that the adductors are implicated only later on." Now this is undoubtedly correct, as shown by experience; and it is acknowledged that Rosenbach's publication appeared a little before my own first special contribution to this question. But with every effort at preserving an objective attitude, I cannot characterise Rosenbach's statement otherwise than as a mere *contention*, most venturesome at the time, and which, luckily for the author, ulterior events proved to have been correct. I maintain most definitely :—

1. That his *generalisation* from *one single case* was not justified;

2. That his statement refers only to *peripheral paralysis by pressure*, not to *central affections*.

3. That Rosenbach has adduced *no real proof of the general calidity* of his statement, even for this pressure paralysis.

For further details the interested reader is referred to the controversy (40).

The first special paper on this question I published appeared under the title, "Clinical remarks on the proclivity of the abductor fibres of the recurrent laryngeal nerve to become affected sooner than the adductor fibres, or even exclusively, in cases of undoubted central or peripheral injury or disease of the roots or trunks of the pneumogastric, spinal accessory or recurrent nerves," in 1881, in the American Archives of Laryngology, (41) since defunct.

This paper culminated in the *establishment of the fact*—it will presently be seen why I lay special stress upon the italicised words—that in the various progressive *organic* lesions of the motor laryngeal nerves, when the lesion of the affected nerve

had not been from the very beginning equivalent to its complete transverse section, symptoms of paralysis of the glottis-*opener* only had, without exception, been observed at the commencement and often up to the death of the patient; and that after death isolated, or at any rate more advanced degeneration and atrophy of the posterior crico-arytænoid muscle had been found; whilst not a single case had been reported in which under similar circumstances the glottis-*closers* had been the muscles first affected. On the other hand, it was remarkable that in all so-called *functional* motor neuroses of the larynx the *ad*ductors had almost always, if not without exception, been the only muscles affected.

An explanation of these facts *was not* attempted in the paper referred to. It is true that different hypotheses were ventilated which could possibly have contributed to explain the curious fact, and—what I wish to mention more especially in view of Krause's hypothesis—*I myself* already then discussed a very obvious possibility, viz., that the phenomena observed during life might *not* belong to a primary *paralysis* of the *abductors*, followed by paralytic contracture of their antagonists, but to a *spasm of all the muscles of the larynx*, with preponderance of the adductors (43). Any such explanation, however, was absolutely rejected in my paper, inasmuch as neither the existence of a spasm lasting for months and even years, in consequence of direct compression of a motor nerve by a slowly growing tumour *had any analogy in the rest of pathology*, nor above all could the important fact be reconciled with this theory that in these cases the abductor muscles only, or at any rate chiefly, are found, *post-mortem*, *degenerated and atrophic*.

Similarly Morell Mackenzie's (44) hypothesis, viz., that the abductor filaments might possibly be more superficially situated than the adductor filaments, or that the latter might be reinforced from the superior laryngeal nerve, as well as Bosworth's theory of the uniformly *central* origin of paralysis of the abductors, were rejected as untenable, and the conclusion of the paragraph in my paper runs as follows:—

"However, all these hypotheses are open to certain objections, and none of them seems to me to offer a really satisfactory and plausible explanation. But at the same time I do not believe that an *explanation* is the thing we want at present."

"In the face of the comparative novelty of, and of all the difficulties connected with, this question, I would rather believe that the time has not yet come when we can venture to give an *explanation*, but that before all by numerous further contributions,

the *fact* should receive further corroborative evidence, that there is a proclivity of the *abductor* fibres of the recurrent nerve to succumb to pathological influences affecting the roots and trunks of the motor nerves of the larynx."

The fate of this paper has been somewhat peculiar. I do, of course, not complain that it shared the common lot of almost all publications which bring anything new, and that it was at first corroborated from different quarters, and afterwards attacked from others (45). But what is really curious is, that just those two points which I myself look upon—if the word "merit" is here applicable—as the two most meritorious ones of the whole paper, have given rise to misunderstandings and controversies which not only have formed a literature of their own, but continue *up to the present hour*, and so render any agreement upon the whole question extremely difficult. It is therefore absolutely necessary that these two points should at last be definitely cleared up.

The first of them concerns the *title* of my paper, or rather the word "proclivity" used in it. If this word had given rise *from the beginning* to misunderstandings as to what I meant, I should of course, attribute the fault to myself. Even now, I will by no means contend that the word, especially in the German translation, has been a very happily chosen one.

If I had spoken of the greater "vulnerability" or "liability" of the abductor twigs and muscles, the misunderstandings which have clustered and are still clustering round the expression, might possibly have been either entirely avoided, or at any rate, been more limited in number. But the strange thing is, that *during the first three years* after the publication of my paper *everybody* understood the expression exactly in the sense in which I myself intended to use it! The proceedings of the Laryngological Section of the 9th International Congress in 1881, in which the whole question was fully discussed by the most competent judges (46), the first confirmations of my observation [see footnote 45], nay, even the first *attacks* on the general validity of my statements,—all show that at first at any rate everybody knew exactly that I meant to signify by the word "proclivity" nothing else but the greater "vulnerability." It was only since 1884 that a change occurred in a number of contributions to this question, as if a sudden belief had arisen that this expression involved some entirely new mystic element; and since then several authors, when they speak of my paper, never quote the word "proclivity" otherwise than between inverted commas.

How I came to use this term is evident from my foregoing description. I had, when I first mentioned the fact in the German edition of Mackenzie's work, used the expression "this greater proclivity (Geneigtheit) of the abductors, to become affected long before the adductors, often even exclusively, is very curious indeed." When about one year afterwards I wrote my first paper on this question in English, nothing was more natural than that I should have simply translated the German expression "Geneigtheit," which I had originally used.

Thus originated the expression "proclivity," and that I should have used in the title of my paper such a still restricting expression, is to be attributed only to a *degree of caution* which indeed I should like to put to my account as a sort of small merit.

I had after ample personal experience, and after a search through the whole laryngological literature, arrived at the result, that in the cases in question so far *without exception* the abductors had been found affected exclusively, or at any rate, more so than the adductors; it would then have been natural enough, as Rosenbach indeed has done *after one single observation*, to formulate this result *as a pathological law*, and to give to my paper such a title as "On the law determining the order in which the individual fibres of the recurrent laryngeal nerve successively are affected in progressive organic lesions of the motor laryngeal nerves." But it must not be forgotten that all this, at the time when my paper was published, was not merely *new*, but was in *direct opposition to the generally accepted doctrines!* I did not wish to incur the possible risk of seeing a few weeks after the publication of my paper, the law I had just proclaimed impugned on the strength of an unimpeachable case. Thus I preferred to be still cautious, and to speak only of a "proclivity," even when the idea of the "law" was before my own mind. That this chain of thought is not a subsequent interpretation, but actually guided me already in 1881, is shown by the following sentence of my first paper (47); "the laryngological literature is already so large that it is not impossible that a few cases of this sort (viz., of initial paralysis of the *adductors*) might have been described, of which I have no knowledge;" and from the fact that to this sentence I make the following foot-note: "I earnestly hope that this paper will serve in eliciting contributions corroborating or refuting, as the case may be, my statements, from those who have had and who have the opportunity of seeing cases in point."

After this full explanation of my chain of thought I trust we may hear no more about the "mysticism" with which my language has been charged.

Still more strange mistakes, however, are made concerning the second point, to which reference was made just now.

From the quotation of the conclusion of my paper it is seen that no *attempt* had been made in it at an original explanation of the newly found phenomena; on the contrary it had been explicitly stated that such an explanation for the time being was not urgently required.

The cause of this self-restriction was simply that the previous attempts at explanation mentioned in the paper were obviously insufficient. A satisfactory hypothesis was prevented by a fact hitherto only casually referred to by Schech (*loc. cit.*), to which my attention had been directed whilst studying the question of abductor paralysis, when I was compelled to observe accurately the position of the vocal cords during quiet respiration. I found that the usual description of the "rhythmical excursions" of the vocal cords during normal respiration *did not apply in the great majority of cases* of healthy adults, in about four fifths of whom the glottis forms an isosceles triangle, which during inspiration is but slightly, if at all, increased, and in expiration is just as little diminished in size. Exact measurements by means of graduated mirrors showed that this triangle was *two-and-a-half to three times larger* than that formed by the glottis *after death* ("cadaveric position" of the vocal cords.) This greater width of the glottis during life, which was not easily explicable *primâ facie*, certainly seemed to point to a *greater strength of the abductors* in comparison with the adductors, and the fact that these apparently *stronger* muscles should more easily succumb than the adductors to organic lesions became the more mysterious. Since then I have persevered in giving attention to this question, and have supplied a solution, I trust, of a satisfactory nature in a paper (48) which I brought before the Royal Society in 1890. In suggesting that the position of the cords during quiet respiration is the consequence of a *reflex tonus* of the posterior cricoarytænoid muscles, I find myself in accord with Krause, who simultaneously with, and independently of me, discussed this question in 1884, on the occasion of the eighth International Medical Congress at Copenhagen (49).

At the time, however, when my first paper on abductor paralysis was published, I had not yet arrived at a full comprehension of the conditions in question, and for that reason *refrained from any* attempt at explanation. That I did so *intentionally* is proved—quite apart from the quotations given above—by the following passage of my second paper on the subject

published in 1883 (50). "An explanation of my own of the fact which here was proven for the first time, I do not give, for the good reason that I do not know of one. Thus much was clear to me, that every attempt to explain the central and the peripheral cases by *different* theories, whilst in both cause and effect were identical, was fundamentally hopeless; to the only common and plausible explanation however, which was before my mind, a certain physiological fact did, at the time, appear to oppose itself as an insurmountable obstacle. Further study of this fact has *now*, I believe, enabled me to understand the real meaning of the pathological phenomenon, and I hope to give the explanation in the course of the forthcoming winter."

These repeated statements seem to me so very unambiguous that it is really quite unintelligible to me how, in spite of them, a number of authors of different nations—and in spite of repeated protests on my own part—have persisted in speaking of "Semon's theory." One can hardly open a paper which deals with the question of abductor paralysis, without encountering such expressions as: "La théorie de Semon," "die Semon'sche Hypothese," "teorema Semon," "Semon's hypothesis," &c., &c. One author summarises my views as follows: "Semon seeks the cause of this curious phenomenon in a proclivity of the abductors to become diseased." Another writer constitutes himself the advocate of Nature, which he thinks I have most seriously offended by attributing to it a "proclivity to attack one of the most vital muscles of the human system." A third admits, it is true, that I had directly stated that I would not attempt to explain the phenomenon, but at the same time outdoes his predecessors by declaring that I had "unintentionally given an explanation by registering the morbid process through the title of my paper under one exclusive cause."

A similar obstinacy in forcing upon an author various theories and explanations, which he not only has never brought forward, but against which he has *expressly protested* in the plainest terms, constitutes a rather exceptional phenomenon in medical literature. It can only be surmised that the authors who avail themselves of such expressions in discussing my papers have either not read them in the original, and have derived their knowledge only from inaccurate or incomplete abstracts, or that their ideas of what constitutes a "theory" and what a "fact" are somewhat hazy.

Anyhow my reply is as follows:—If during life in a case in which the *whole* of the nerve trunk or the *whole* centres of the motor laryngeal nerves on one or both sides are organically

affected, I observe the symptoms of paralysis of one or of both the glottis-openers, and this under circumstances which *everywhere else in the whole range of pathology* would induce every observer to think of *paralysis* only and not of *spasm;* and if *post-mortem* I find the posterior crico-arytænoid muscles shrunk to hardly recognisable discoloured bundles, whilst the adductors, which are supplied by the same nerve, are either normal or at any rate much less degenerated and atrophied, I then establish a "fact," but I do not evolve a "theory," by stating that in this case the abductors have given way before their antagonists. If this occurs not only in a certain number of cases, but *without exception* in all cases of various *organic lesions progressively affecting* the whole centres or trunks of the motor laryngeal nerves, then the law necessarily follows that the nerve fibres of the motor laryngeal nerves going to the abductors, and these muscles themselves, succumb to organic affections sooner than, or even exclusively of, the adductors.

This is what I have stated, *this* is what up to the present day has remained a positive fact, and to speak of it as a "theory" or a "hypothesis" is inadmissible.

My language may appear too emphatic to those who are not conversant with the literature of this question, and it may possibly be objected that this interferes with the objectivity of a historical description. I felt it, however, to be my duty to give such a description of the development of these questions as will, I hope, be useful to future investigators; and to refer in plain language in the hope of removing them, if possible, to the truly incredible (51) misconceptions, which up to this day stand in the way of an agreement between the adherents of the different views.

My last-named paper formed the starting-point, so to say, of a new branch of laryngological literature, which, scanty from 1881 to 1883, increasing very considerably during the following three years, attained its maximal development about the year 1886, and which has since grown so as now to fill nearly three large volumes.

The character of these contributions varies very considerably. In the first place numerous casuistic communications have been and are being contributed, confirming the fact stated by me. Amongst these I may mention the papers by Schäffer (52), Jonquière (53), Kothe (54), Ziegelmeyer (55), M'Bride (56), Dehio (57), Bristowe (58), de Havilland Hall (59), Garrod (60), Percy Kidd (61), Gordon Holmes (62), J. Israel, Remak, B. Fraenkel (63), Aronsohn (64), Schech (65), Landgraf (66), Martius (67), Dreyfuss (68), Neumann (69). To the same category belong also

the commentaries of Riegel (70), Massei (71), Buzzard (72), Ormerod (73), Ferrier (74), Gottstein (75), Ariza (76), acknowledging the correctness of the fact. Some of these authors (Buzzard and Ormerod) endeavour to utilise and explain analogous pathological occurrences in other organs, as for instance the order of events in the disablement of the individual muscles of the eye.

We now come to the interesting observations by Jeanseline and Lermoyez (77), on the dead bodies of cholera patients, showing that of all the laryngeal muscles supplied by the recurrent laryngeal nerve, the posterior crico-arytænoids are the first to lose their electric excitability after death—an observation which Horsley and I (78) confirmed in various species of animals. The fact also, corroborated by Fleming (79) and Clarke (80), that in the so-called roaring of horses, which Fleming attempts to trace mainly to the pressure upon the left recurrent laryngeal of enlarged lymphatic glands, situated near the arch of the aorta, the left abductor is always found to be degenerated alone or chiefly so, points in the same direction.

In opposition to this evidence, attacks upon the general validity of my statements were made at first in the form of publication of isolated cases, which in the opinion of their authors appeared to prove that in organic progressive disease of the motor laryngeal nerves the adductors occasionally become affected first. To this category belong the papers by Schnitzler (81), S. Solis-Cohen (82), J. Solis-Cohen (83), and Charazac (84). It was easy for me to show in my second (85) paper, published in 1883, that the cases published by Schnitzler and J. Solis-Cohen do not in the least prove the assertions made by their authors; and the same may be said of the more recent cases published by S. Solis-Cohen and Charazac. The first of these is in all probability of the nature of a *mechanical* disturbance of mobility; the second most likely of a *local* disablement of one or the other of the adductors. These cases have not been quoted by anybody but by their authors as invalidating my law. The very interesting cases published by Remak (86), (Bilateral paralysis of the spinal accessory nerves), Cartaz (87), (Cases of laryngeal paralysis of central origin), Seifert (88), (Saturnine paralysis of the larynx) can be cited neither for nor against my statements.

With reference therefore to any attempts to refute my law by *casuistic* proofs, I must now emphasize the following statement:—

Whilst a large number of cases of progressive organic disease of the motor laryngeal nerves have been published, in which the

abductors have been shown to have undergone, either alone or at any rate much more than the adductors, fatty degeneration and atrophy (89), *not a single specimen has been hitherto demonstrated, which, under similar conditions, exhibited the opposite order of events in the development of degenerative changes in the individual laryngeal muscles !*

All such attacks made so far upon the law exclusively rest upon *clinical* observations, which are either incomplete or are capable of an interpretation different from that adopted by their authors.

The theoretical doubts of Gouguenheim (90) and Paul Koch (91) need only be referred to in passing. Gouguenheim and his followers, who consider the median position of the affected vocal cord to be a spastic phenomenon, believe that the latter is due in a large number of cases to pressure of the peritracheo-laryngeal ganglia, described by himself and Leval-Piquechef (92), upon the recurrent laryngeal nerve.

Shortly after the commencement of the period of casuistic corroboration and attacks, the first attempts at explanation were made. Curiously different hypotheses were brought forward in order to render more intelligible the clinical phenomenon. Thus Gowers (93) thinks that it may possibly be a consequence of the advantage with which the most important adductor, the lateral crico-arytænoid muscle, works in comparison with the abductor (in so far as the former goes in a right angle, the latter in a very acute angle towards the muscular process of the arytænoid cartilage), which renders the adductors capable of longer resisting disabling influences which affect the whole nerve trunk.

W. Robinson (94) attempts to explain the fatty degeneration and atrophy of the abductors, which had been observed by myself (95) in a case of goitre, by a supposed disturbance of their vascular supply, inasmuch as the posterior crico-arytænoid muscles are supplied by the inferior laryngeal arteries which had probably been compressed in this case; whilst the immunity of the adductors was, he thought, to be explained by assuming that the superior laryngeal arteries which supply them had not been injured by the pathological process.

The third hypothesis is due to Cohen-Tervært (96), and is founded upon the results of Exner's (97) well-known paper on the innervation of the larynx. It may here be mentioned that I shall not further refer in this historical sketch to Exner's paper, nor to the studies of his pupils, Mandelstamm (98) and Weinzweig (99), interesting and meritorious as they are, because there is, so far

as I can see, no direct connection between them and our present subject, and because they have led to no results of any practical value.

The distribution of the motor nerves to the individual laryngeal muscles, as stated by Exner, and the anomalies of nerve distribution occurring everywhere, led Tervært to imagine that in the cases now discussed, the nerve-supply of the laryngeal muscles deviated from the rule in so far that the abductor is innervated by the recurrent laryngeal, to the exclusion of the superior laryngeal nerve; whilst the other muscles received more motor fibres from the superior laryngeal nerve. This hypothesis, which is based upon the assumption that in cases of abductor paralysis in which the entire nerve trunk is affected, the isolated paralysis of the posterior crico-arytænoid muscles must necessarily persist to the very last, collapses before the simple fact that in many cases, the initial abductor paralysis finally evolves into complete recurrent paralysis (100).

Further, if this hypothesis were correct, in the cases in question the external thyreo-arytænoid muscle ought always to be found to be at least as much atrophic and degenerated as the posticus. For, whilst, according to Exner, the latter is supplied only *predominantly* by the recurrent laryngeal, according to the same authority, the external thyreo-arytænoid is "usually *exclusively*" innervated by the recurrent. Such a condition of things, however, has not been found in any of the numerous cases in which abductor paralysis has come under observation. Many more objections could be raised against Tervært's hypothesis, but these two suffice to show its untenability.

The complex far-fetched and not very intelligible theories brought forward by S. Solis-Cohen (101) and Jelenffy (102), the mere statement of which would demand a disproportionate amount of space are so little founded upon facts, that they are only alluded to here for the sake of completeness.

Tissier's hypothesis is equally shadowy; the recurrent laryngeal is not a simple nerve, but contains (i.) vagus fibres, (ii.) spinal fibres. The former are said to be present only in small numbers in the recurrent laryngeal, and their excitability is supposed to be lost sooner than that of the spinal fibres. In compression of the nerve or in peripheral neuritis, the vagus fibres are thus the first to be exhausted, the spinal fibres retaining their function, which being phonatory allows the approximation of the vocal cords.

A very curious hypothesis has been quite recently proposed

by Wagner (104) of Halle. This author found that after unilateral as well as after bilateral section of the recurrent laryngeal nerves in cats, and in the very few dogs experimented upon, the corresponding vocal cords assumed a median position and immobility which lasted for a few days and then passed over into the so-called "cadaveric" position. He explains the median position of the vocal cord or cords under such circumstances as being due to the action of the *crico-thyroid muscle only*.

This explanation appears so simple that it would be extraordinary if no previous observer had found it, provided, of course, that the author's facts are correct.

Now it would appear that Wagner is not aware that section of the recurrent laryngeal or laryngeals has been performed before him by *numerous* experimenters; at least he does not allude anywhere to the analogous experiments of his predecessors, nor does he attempt to explain the differences between his own and their results. Had he studied the literature of the subject he would have seen that experiments concerning this question have been made since the beginning of the present century by Legallois, Read, Longet, Traube, Rosenthal, Dalton, Georg Schmidt, Schech, Steiner, Vierordt, jun., Horsley and myself, &c., and that although all these observers agree that after section of the recurrent laryngeals the glottis becomes much narrower than it had been previously, yet that there are very great differences as to the *degree* of the narrowing, according to species, age and individual peculiarities of the animals experimented upon. While certain species, such as cats and horses, not only in the first days after birth, but even when adult, are suffocated by the section of the recurrent laryngeals, other species, notably dogs, suffer less and less, in proportion to their age, so that, whilst they die when operated upon a few days after birth, dyspnœa only occurs on exertion when they are operated upon when adult. Again, other species, such as rabbits and guinea pigs, are not nearly so much inconvenienced when operated upon, even at a very early period of their existence. The reason of these differences is well explained by Legallois, the first thorough worker in this field (*Nouveaux éléments de physiologie*, 2 Edit., vol. xi., p. 326 et seq.), as follows:—

"It rests upon the fact that, in proportion to the capacity of the lungs, the opening of the glottis in animals of the same age is greater in one species that in another, and still greater in the adult than at the moment of birth in the same species, as already stated by M. Richerand, in the human species. Or, assuming

that the form of the glottis on the whole is similar in these diverse animals, inasmuch as the areas of the smaller figures are to each other as the squares of the homologous dimensions, it is seen that a narrowing of the same kind of the glottic opening must intercept the passage of air in very different degrees."

From this quotation alone it is evident that there cannot be in all cases bilateral median position of the vocal cords, *i.e.*, *complete closure* of the glottis, after section of the recurrent laryngeals, for, if such were the case it would be impossible to understand how any animal thus operated upon could have *survived* the operation. Yet Legallois and many of his followers have kept such animals alive for long periods. Moreover, several of the experimenters, who directly *inspected* the position of the vocal cords after section of the recurrents, *e.g.*, Traube, Schech, Steiner, Horsley and myself, Katzenstein, have actually *seen* the corresponding vocal cord recede into the *cadaveric* position at the *very moment* its recurrent laryngeal nerve was cut.

This is entirely at variance with Wagner's results, who, without exception, found in his unilateral sections, the median position of the vocal cord to last up to six days. Yet he does not as much as mention this grave discrepancy between him and his predecessors.

Now it appears to me to be the *duty* of any observer who enters upon a field well trodden by previous investigators to examine their *facts*, if not their *theories*, and either to refute them or to reconcile with them any new hypothesis which he, as the result of his own experiments, may bring forward. But he has no right, I think, to leave such facts simply out of court, and to give an entirely erroneous impression of the actual state of matters to readers who may not be familiar with the subject. This is what Wagner has actually done.

Since his publication Katzenstein, of Berlin, has made a series of very careful and varied control experiments, and has been unable to corroborate Wagner's statements; his own results, which are given in Virchow's Archiv, vol. 128, entirely agreeing with those of the older observers.

This being so I shall confine myself to pointing out a few other obvious objections which might be raised against Wagner's *interpretation*, assuming for a moment, what I do not admit, that his *facts* are correct.

1. Supposing that after mere section of the recurrent laryngeal nerve the vocal cord at once *invariably* went, as he describes it, into the middle line, and that this was due, as he thinks, to the

action of the crico-thyroid muscle; it necessarily follows that the position of the vocal cords, which we see in quiet respiration in man, is not merely a result of a reflex-tonus of the abductors (as would appear from my paper in the Proceedings of the Royal Society, 1891), or even of a simultaneous innervation of the abductors and adductors, with preponderance of the former, but that the *crico-thyroid* also must play an important rôle in the production of this position. Surely the author cannot believe for a moment that this muscle only *began* to exercise a constant activity at the very moment at which the recurrent laryngeal was cut? Such a belief appears so inherently improbable as to be not worth discussing. Supposing, however, on the other hand, that the crico-thyroid muscle in man is *always* engaged in maintaining the position of the vocal cords during quiet respiration, and in counter-balancing, to a certain degree, the action of the posterior crico-arytænoid muscle (as we must conclude from Wagner's results from recurrent section), it would again follow that, if the *crico-thyroid* itself were disabled, the glottis in quiet respiration ought to be *wider* than it is under normal conditions. Now I am not acquainted with a single *pathological* observation pointing this way, whilst in *experiments* in which the crico-thyroid has been cut in animals (*e.g.*, by Schech and Steiner), no appreciable change in the width of the glottis was observed. Neither did Katzenstein obtain any widening of the glottis after dividing the external branch of the superior laryngeal nerve which supplies the motor filaments to the crico-thyroid muscle. Thus neither experiment nor pathological observation give the least support to Wagner's contention, whilst he himself never once enters upon the all-important point I have just raised.

2. Wagner, in the whole of his three extensive papers on the subject never condescends to investigate the cause of the exclusive or more developed degeneration and atrophy of the posterior crico-arytænoid muscle in cases in which median position of the vocal cord from progressive organic paralysis of the recurrent laryngeal nerve had existed during life. This point he does not even mention, though he himself reports in several of his experiments that the electric excitability of the posterior crico-arytænoid was lost after death previous to that of the adductors.

3. Finally, no explanation whatsoever is given of the fact that the median position in man may and does last for many years, whilst its maximum duration in Wagner's experiments was six days.

More might be said about the defects of the author's bibliographical researches, about the probable sources of fallacy in his experimental methods, about his confident generalisation of the results of a few experiments, and about his own far-fetched explanations; but what we have just seen will be enough to show that in spite of their apparent simplicity, Wagner's views are not likely to meet with general acceptance.

Having so far disposed of the theoretical, we now reach the anatomical and histological contributions. Grützner (105) appears to be inclined to look upon the adductors as "white," and the abductors as "red" muscles; Simanowsky (106), who worked in Grützner's laboratory, directly characterizes them as such. These results are opposed to the fact which may now be considered as certain, viz., that the abductors first succumb to any organic progressive lesions of the whole nerve, and to the physiological experiments which have been made by Horsley and myself (107); and I must content myself here with pointing out these as yet unsolved contradictions. If in reality the abductors belonged to the class of red, the adductors to the class of white muscles, we should have expected, in accordance with what we know of the biological properties of these tissues, the abductors to resist organic pathological influences longer than the adductors.

We shall presently see that this bio-chemical question has to be diligently studied, as it probably contains the final explanation of the different resistance of the glottis-openers and glottis-closers to progressive nerve-lesions.

Finally, we must here mention the experiments concerning the excitability of the recurrent laryngeal nerve, made by Franklin H. Hooper (108) and Frank Donaldson, Junr. Hooper's remarks in the discussion which followed the reading of his paper (*Transactions of the American Laryngological Association*, 1885, pp. 23 et seq.), probably contain the most violent attacks ever made upon the fact of the greater vulnerability of the abductors. But they are not based upon valid data, nor do they in the least disprove the fact stated by me. The real meaning of the important discovery which Hooper accidentally made in the course of his experiments, viz., that ether possesses a peripheral and differential action upon the laryngeal muscles, was not duly appreciated by the discoverer himself, and was recognized subsequently and explained by Horsley and myself (110).

In the first of his papers Donaldson directly attacks the fact of the greater vulnerability of the abductor fibres; in the second, however, he fully acknowledges it. These contributions possess

permanent value as demonstrating the greater excitability of the abductors when weak electric currents are used. Both the last-named authors have followed up their original observations by further interesting excitation experiments on the recurrent laryngeal nerve; but their later papers do not appear to shed any special light upon laryngeal paralyses.

We thus see that there has been no want of diversity among the various hypotheses propounded in explanation of the phenomenon of the greater abductor vulnerability, although none of them has succeeded in gaining general acceptance. The majority have only raised a passing interest, the rest being still-born.

The last hypothesis to be now mentioned, however, has rallied a number of adherents, whilst others are maintaining an expectant attitude in the fight between its partisans and those of the older view. I am alluding to Krause's contracture hypothesis (111).

Krause started with the wish to imitate by experiment the pathological process, a sufficient explanation of which at that time had apparently been given neither by clinical observation nor by the results of autopsies, but failed in a preliminary series of experiments to produce the laryngeal phenomenon, as it were, under his eyes. He now attempted to imitate experimentally the pathological process upon which the median position of the vocal cords most frequently depends during life, viz., the pressure of a tumour upon the motor nerves of the larynx. With this view he fixed a piece of cork, by means of an indiarubber band or a violin string, softened in water, to the previously carefully isolated recurrent laryngeal nerves, which were then replaced in situ. After a few hours he first observed slight vibrating twitchings, afterwards somewhat temporary median position, and after about 24 hours, lasting median position. This median position persisted without any change for two or three days. Afterwards it passed over into complete paralysis. If the same experiment was performed on the pneumogastric nerve (it deserves special mention that Krause always operated on both sides), the vocal cords assumed permanently the quiet position of expiration.

From these experiments Krause concludes that the phenomenon produced by him must be looked upon as one of *excitation*. He attempts to prove, partly by a few clinical cases which had been previously reported, partly by theoretical considerations, partly by quotations from the writings of neurological authorities, that in *man* also the pathological process does not consist as hitherto assumed, of a *primary paralysis* of the posterior crico-arytænoids, followed by paralytic contracture of the antagonists,

but of a *primary* neuropathic *contracture* of all the muscles supplied by the recurrent, with preponderance of the adductors.

The above is a faithful, though concise, analysis of this important paper. The reader must for details refer to the original.

It is not surprising that Krause's theory should have found and still finds numerous adherents. It provides an intelligible explanation for the previously obscure cause of the phenomenon; it is *prima facie* very attractive, owing to its great simplicity; the few experiments on animals on which it rests, are considered by some of greater value than any series of careful clinical and pathological-anatomical observations; finally, it must undoubtedly be conceded that Krause's paper ably sustains his views.

Thus a number of laryngologists have declared themselves partisans of this theory, amongst whom are Möser (112), Gouguenheim (113), J. Solis-Cohen (114) (if I correctly understand his present views), Moure (115), and lastly Massei (116), who has declared himself a convert.

Now the weak points of Krause's theory are so obvious, that from the moment of its publication it encountered opposition. The author having first reported the results and the interpretation of his experiments to the Physiological Society of Berlin (117), submitted them again to the Laryngological Section of the eighth International Medical Congress at Copenhagen (118). In the discussion which followed his paper (*l.c.*) B. Fränkel, Boecker and myself raised objections against his views. I declared (*l.c.*) that whilst reserving my judgment concerning the general idea in support of which these experiments were adduced, the following objections at once came up to one's mind:

1. In cases of *multiple* paralyses, such as had been described by Remak and myself, is the affection of the vocal cords which is present amongst others signs, also to be looked upon as a contracture? Why amongst a number of undoubted paralyses this one contracture? 2. The occurrence of so complete a degeneration of the abductors as is found in these cases as a pure consequence of the contracture of the adductors was unintelligible. 3. It would be in direct opposition to *all pathological experience*, if organic disease of the vagus should produce lasting expiratory position of the affected vocal cord, as seen in Krause's experiments. On the contrary in all such cases, except when the paralysis was complete, one observed unilateral or, in some cases, reflexly produced *bi*lateral *abductor paralysis*. I added that other objections presented themselves, but that considering the difficulty

of the question and of the experiments, we must wait for the full publication of Krause's paper.

This paper having been published, O. Rosenbach (119) and B. Fränkel (120) were the first to raise further objections against Krause's views. Both authors first criticised the experiments themselves, and next the interpretation of their results as a contracture. Möser and Tervært (*loc. cit.*), also brought forward various criticisms of Krause's interpretation. To Rosenbach and Möser's objections Krause replied in two short papers (121) of a polemical nature, the second of which finally cleared up the question as to *which* muscles were to be considered as being in a condition of contracture. It must be explained that up till then repeated misunderstandings had been caused by Krause's varying modes of expression. Sometimes one could not but imagine that he fancied that only the "*main bulk*" of the laryngeal muscles, *i.e.*, the adductors, were in a condition of contracture, sometimes he spoke of a contracture of *all* muscles supplied by the recurrent laryngeal. His reply to Möser finally settled matters definitely in that sense that in his opinion *all* laryngeal muscles supplied by the recurrent were contractured.

Gerhardt (122), Michael (123), and Weil (124), about the same time, published papers which took an intermediate position between the old and the new views.

My own objections against Krause's theory were brought forward as late as 1886 in a paper entitled: "Abductor Paralysis, not Adductor Contracture" (125), which was read before the Laryngological Section of the German Naturalists' Meeting at Berlin. I had considered it necessary, in view of the surprising statements which Krause had made about the position of the vocal cords in experimental compression of the pneumogastric nerves, to repeat the experiments themselves. This occupied much time. My paper forms part of a larger work, the MS. of which already in 1886 was nearly ready for the press, but which for various reasons has up to the present not been published. Of the paper itself therefore, only short abstracts in the Journal of the Naturalists' Meeting, and in a few reports of the proceedings have been published; and this is probably the reason why my arguments against the contracture hypothesis have apparently not become so generally known as my first articles and as Krause's own paper. I hope soon to publish the whole work; but will give here a full abstract of my paper, which contains my main reasons for not accepting Krause's theory; reasons which, so far as I know, have up to the present day remained entirely unrefuted.

After protesting against the validity of cases, observed *clinically* only, as counterproofs to my law, I proceeded by giving a report of several series of experiments on different species of animals undertaken by Mr. Victor Horsley and myself conjointly. From these it appeared that:—

1. The abductors are the first of all the laryngeal muscles proper to lose their excitability after death.

2. After thrusting a thread saturated with chromic acid solution through a recurrent laryngeal nerve, the animal is killed a week afterwards; the corresponding posterior crico-arytænoid muscle is the *first* to lose its excitability.

Here I noted, first, that I had not been able to corroborate, in man, Krause's statements as to the complete closure of the glottis during cadaveric rigidity; secondly, that the expression "cadaveric position of the vocal cords" was open to objections, inasmuch as the position of the cords after death varied considerably.

I then discussed at some length the sources of error attending experiments upon animals made for our purpose, and of which I adduced not less than eight, met with by ourselves. I drew from my experience the conclusion that experiments on animals can here be utilized only with the greatest precaution, and can certainly not be regarded as *decisive*.

If this were generally true as regards physiological experiments performed on the laryngeal nerves, it was still more so in the case of experiments intended to be *imitations* of usually *chronic* pathological processes. Krause's conclusions were, in my opinion, too far-reaching, mainly because he had identified the *sudden* and *intense* irritation he had experimentally produced, with the slow and *gradual* increase of irritation by chronic pathological processes. That the median position produced by him experimentally was to be interpreted as an *irritative* phenomenon, I quite admitted, although there was no need to term it a "primary neuropathic contracture" (this point was fully discussed); nor did I deny that in *a few* human cases of *acute* character a neuropathic median position of the vocal cord might have to be interpreted as an irritative phenomenon. It was undoubtedly meritorious on Krause's part to have drawn attention to this point, but I must reject such a *generalisation* as that contained in his papers. In man a *slow* destruction of the nerve almost always takes place, in which alterations of pressure, and with them irritative phenomena, *can* no doubt occur, but are *very frequently* absent. Even in cases of this kind the posterior

crico-arytænoid muscles, as shown by the result of the autopsy, succumbed first. I showed that Krause fell into self-contradiction when interpreting the atrophy of these muscles, as "atrophy due to inactivity," since according to his hypothesis *all* the muscles supplied by the recurrent laryngeal, and therefore also the abductor, were supposed to be in a condition of chronic irritation.

The following consideration, moreover, completely settled the question for me. If Krause's general conception of the nature of the processes taking place in these affections of the recurrent laryngeal nerve were correct, it would logically follow that *everywhere in the system where absolutely analogous processes occurred under absolutely analogous conditions the same result must uniformly take place*. Therefore, wherever pressure exercised by a slowly growing tumour gradually disables a motor nerve, a contracture must uniformly ensue. But we never find contracture in such cases, but *paralysis* (*e.g.*, paralysis of the facial nerve or of the brachial plexus, crutch-paralysis, &c.)

This was decisive for me. The quotations from the works of Charcot, Erb, Eulenburg, Hitzig, Seeligmüller, by which Krause attempted to support his views were not admissible to solve the question at issue, inasmuch as they referred to entirely different conditions. These authors had themselves stated so much to me in reply to definite questions addressed to them on the subject.

Horsley's and my own experimental results with reference to the vagus *entirely* differed from Krause's, whilst they tallied, so far as they went, with the conditions met with in pathological cases.

Finally, if further corroboration were needed that the neuropathic median position of the vocal cord or cords was to be looked upon as the result of a primary abductor paralysis, we found it in cases of *multiple paralyses of cranial nerves*, such as had been accurately observed by M'Bride and by myself among others.

In such cases the domains of *all other* affected nerves displayed nothing but *paralytic* phenomena. It was most improbable that *irritative* phenomena should always occur in the domain of the vago-accessory, whilst in all other nerve-territories *paralytic* symptoms should be the rule.

Krause, no doubt, deserved our thanks for having emphasized the possible occurrence of irritative phenomena; and opening our eyes in *acute* instances of neuropathic median position of a vocal cord or cords, to the contingency not only of abductor paralysis but also of acutely irritative lesions, but the *general* interpretation of the phenomenon as contracture was inadmissible, and the

usual soundness of the old interpretation of the median position, as abductor paralysis was unimpaired.

In the discussion which followed my paper Krause defended his interpretation and criticised Horsley's and my own experiments, but admitted that in nerve affections phenomena of irritation as well as of paralysis could occur, and suggested that it might perhaps have been the enthusiasm of the investigator, which had made both him and myself too far exclusive, each of us saying: only one thing of the two can be true, the one excludes the other.

The result of the discussion was to the effect that *not one* of the numerous speakers who took part in it shared Krause's views; Remak, from the general neurological point of view, stated that he entirely agreed with me in thinking that such difficult problems are generally insoluble by means of rough experiments, and can only be explained by clinical and pathologico-anatomical observations, and that laryngologists, on the strength of some quotations, were not entitled to represent as constant a phenomenon in the domain of the laryngeal nerves, which was not observed in the territory of other motor or mixed nerves (120).

This discussion was, at the time when this paper was published in German (October, 1891), the last important occurrence in the question now before us. In 1890, on the occasion of the demonstration of the motor innervation of the larynx, which Horsley and I gave in a combined meeting of the Physiological, Neurological and Laryngological Sections of the Berlin International Congress, I accurately restated my views (127). No opposition was raised, and I believe that even then the opinion of the great majority of laryngologists, and of almost all neurologists was to the effect that in organic progressive lesions of the motor nerves of the larynx the abductor fibres and muscles regularly succumb first.

Since that time, however, some very important contributions have been published. In the first place Risien Russell has materially strengthened in a paper read before the Royal Society, on March 31st of the present year (*Proceedings of the Royal Society*, vol. 51), the position which I have throughout taken in this question, by showing experimentally—

1. That the abductor and adductor fibres in the recurrent laryngeal nerve are collected into several bundles, the one distinct from the other, and each preserving an independent course throughout the nerve trunk to its termination in the muscle or muscles which it supplies with motor innervation.

2. That when the abductor and adductor fibres are exposed in the *living* animal to the dry influence of the air under exactly similar circumstances, *the abductors lose their power of conducting electrical impulses very much more rapidly than the adductors; in other words, that they are more prone to succumb than are the adductors.*

3. That it is possible to trace anatomically the abductor and adductor fibres throughout the whole length of the recurrent laryngeal nerve to their termination in the one or other group of laryngeal muscles, and that these fibres appear to bear a fixed relationship to each other throughout their course, the abductors being situated on the *inner* side of that nerve or next to the trachea, while the adductors are on the outer side.

4. That it is possible to accurately separate these two sets of fibres in the nerve trunk.

5. That the bundle of nerve fibres concerned with one function may be divided without injury to that concerned with the opposite function, and that such division is followed by atrophy and degeneration of the muscles related to that function without any such changes being detectable in the muscles related to the opposite function.

These results, the actuality of which has been demonstrated by the author at a meeting of the Physiological Society, entirely bear out the position taken by me.

In justice to Dr. Onodi, of Budapest, it must be observed here that he communicated results practically identical with those obtained by Russell in the course of the discussion which followed Horsley's and my own demonstration at the International Congress of Berlin, in 1890. But, as he merely communicated the bare facts, without supporting them by satisfactory evidence, his observations could not claim the same value which Russell's careful experiments undoubtedly possess.

Another equally important and surprising contribution to that question comes from the pen of Krause. (*Berliner Klin. Wochenschrift*, No. 20, 1892). On the strength of new experiments on animals he arrives at the conclusion that the recurrent laryngeal nerve is not, as hitherto almost generally admitted, a purely motor nerve, but that it contains also centripetal fibres, and that irritation of this nerve therefore, apart from causing arrest of respiration and of the diaphragm in the expiratory position, also produces narrowing of the glottis and arrest of the vocal cords in the adducted position. This position, therefore, must, in his opinion, now be looked upon as the result of a 'reflex contracture.'

In a very trenchant paper, Burger (*Ibid.*, No. 30), has shown that this new position of Krause's is not, as this author maintains himself, a mere 'modification' of his former views, but constitutes an absolutely new hypothesis, which is additionally quite incompatible with Krause's former theory, according to which the median position of the affected vocal cord was to be looked upon as a 'primary neuropathic contracture.' Burger has repeated Krause's experiments, and has not been able to corroborate his statements concerning the existence of centripetal fibres in the recurrent laryngeal nerve—a result which years ago also Horsley and I have obtained. As Krause, in the course of his paper, admits that the posterior crico-arytænoid muscle may possess the least power of resistance of all the laryngeal muscles supplied by the recurrent nerve, Burger justly sees in this admission a corroboration of the facts for the correctness of which I have contended from the very first, and it may fairly be hoped that this long continued strife is now at last drawing to a satisfactory conclusion.

Nor are we now any longer quite in the dark as to the cause of the phenomenon.

The fact discovered by Hooper (128), controlled and explained by Horsley and myself (129), that ether has a peripheral and differential effect upon the laryngeal muscles, which can be produced only by means of the circulation; the fact that the abductor muscles die sooner than the adductors (Jeanselme and Lermoyez, Semon and Horsley, Onodi, Risien Russell); and the fact demonstrated by B. Fränkel and Gad (130), that gradual cooling of the recurrent laryngeal nerve paralyses the crico-arytænoideus posticus sooner than the glottis closers; all these facts taken together with the clinical and pathologico-anatomical experiences concerning the earlier destruction of the abductors in progressive organic lesions imply that there exists an actual difference in the *biological* composition of the laryngeal muscles and nerve endings, whilst the fact that also in *central* (bulbar) organic affections, such as tabes, the cell groups of the abductors succumb earlier than those of the adductors, points to the probability that similar differentiations exist in the nerve-nuclei themselves. The hitherto obscure phenomenon thus finds its explanation in biological differences between the components of the laryngeal nerves and muscles. This constitutes an *addition* to our knowledge of the nervous morphology, but does not necessitate so *revolutionary* a postulate as that involved in the contracture hypothesis that the motor laryngeal nerve possesses

a *special* pathology, peculiar to itself. We formerly knew that there existed differences between the irritability and power of resistance of the sensory and those of the motor nerves; but assumed complete equality among motor nerves. Now we have also learnt that more subtle differences exist among these nerves and the physiological conditions of the muscles they supply.

Between the years 1880 to 1890 the interest in laryngeal paralyses was concentrated upon organic progressive lesions, from the vago-accessory nucleus *downwards*, and especially upon the abductor-paralyses just described. Recently, however, the question, whether central lesions situated *above the medulla*, especially cortical affections, may also produce organic laryngeal paralyses, has arisen. The pathological aspect of this question has grown out of the modern physiological studies concerning cortical localisations, and which have led to the discovery of definite areas presiding over the phonatory (volitional) functions of the larynx.

Ferrier (131), and shortly after him, Duret (132), in the course of their experiments reached the conclusion that the function of phonation must be represented in the cortex, but did not succeed in obtaining an exact localisation. Krause (133), who studied this question at H. Munk's instigation in 1883, first defined the exact locality of the phonatory area in the dog's cortex. He found that closure of the glottis (*bilateral* adduction of the vocal cords) takes place on stimulating the isthmus ("Stiel") of the præfrontal gyrus (gyrus præcrucialis, Owen). Krause's results were corroborated and extended by Horsley and myself (134). Those results of our investigations are as follows:—

1. That there is in each cerebral hemisphere an area of bilateral representation of the adductor movements of the vocal cords. (Corroboration of Krause's results).

2. That we could discover in none of the species (except cats) of animals experimented upon, an area in the cortex for the abductor movements.

3. That after complete excision of the cortical areas for adduction (stimulation of the neighbourhood of the lesion producing no more effect upon the larynx), and after allowing the wound to heal aseptically, no paralysis of the cords is observed. Further, that on subsequent excitation of the corresponding area in the opposite hemisphere a bilateral adduction of the cords is produced as completely as if the opposite area were intact.

From these facts we argued as follows: "Unilateral destruction produces no effect. There is, therefore, no such thing as unilateral paralysis of a vocal cord from lesion of a cerebral hemisphere, as has been asserted. The truth of our statement is made evident by the fact that motor aphasia is not identical with aphonia. Moreover the observation by one of us (F.S.) of several persons with complete aphasia and right hemiplegia only a few hours after an apoplectic stroke, has conclusively shown that in such instances the movements of the vocal cords are perfectly preserved."

Mott (135a) and Sherrington (135b) corroborated these conclusions, but François Franck (136) and Masini (137), arrived at very different results.

The former denied the existence of any differentiated representation of the larynx, or of other parts of the respiratory apparatus, in the cerebral cortex. Krause (138) has sufficiently answered this statement. Masini found in four cases, that *weak* stimulation of the phonatory cortical area produced at first isolated movements of the opposite vocal cord, followed by weaker movements of the vocal cord corresponding to the stimulated side. He is, so far as I know, the only experimenter who has obtained this result; in spite of the large number of controlling experiments concerning this point which Horsley and I have undertaken, both before and after the publication of Masini's statements, we have not been able once to corroborate them. Yet this is just the crucial point, whether *unilateral* cerebral lesions can produce organic unilateral laryngeal paralysis.

Although a certain number of cases have previously been published (139), (Andral, Gerhardt, Friedreich, Foville, Duval, Löri, Lewin, Livio Ronci, Luys, Cartaz, Massei, &c. (140), in which vocal disturbances or even laryngeal paralyses have been observed simultaneously with organic lesions of the higher centres, they cannot be utilised towards deciding the present question because they are all, in some way or other, incomplete. In some the diagnosis of a cerebral lesion is doubtful; in others multiple lesions exist; in a third category no laryngoscopic examination has been made; in others, again, either no autopsy was made or was described in a defective manner with regard to the condition of the medulla, the nerve fibres and muscles themselves.

In 1884 Bryson Delavan (141), called attention to what he considered as a possibility of localising, from clinical observation, the laryngeal cortical centre. Of the two cases, however, upon which he bases his views, in one (Seguin) no laryngoscopic

examination had been made; and he himself confessed (142) some years afterwards, that in his second case the laryngeal paralysis, supposed to be due to a cortical lesion, was of bulbar origin, as shown by the *post-mortem* examination.

Rossbach (143) has recently published an interesting paper, in which, from a carefully observed case, he draws the conclusion that unilateral paralysis of a vocal cord may be caused by lesion of the opposite insula. This is, however, invalidated by the considerations that there was atrophy of the corresponding side of the tongue, due to an undoubted bulbar lesion of the hypoglossal nucleus; that the integrity of the vocal accessory bulbar nucleus was merely *surmised* from the fact that the fibres of the nerve itself and of the laryngeal muscles had undergone no atrophy; and that he did not prove that the laryngeal paralysis had existed so long that atrophy must necessarily have followed.

In a recent paper Scheff (144) throws no light upon the pathogenesis of his cases; but the controversy between Messrs. Garel and Dor on the one hand, and Professor Horsley and myself on the other, which followed (145) the publication of a case of the French authors just named (146), is of importance for us.

Garel had previously reported (147) a case, from which he concluded that a unilateral laryngeal paralysis may be caused by cortical lesion of the opposite hemisphere. Horsley and I (148) at the time took exception to this view on the basis of our experiments.

Garel, in his paper with Dor, maintains his original view, and on the strength of the autopsy expands it into the statement that a unilateral lesion of the internal capsule also can produce *complete* paralysis of the vocal cord of the opposite side. To this interpretation Horsley and I made the following objections:— 1. In this case, according to the authors' own report, the medulla has not been *microscopically* examined, and therefore the absence of a lesion of this part is absolutely unproved. 2. The *complete* paralysis of the vocal cord (according to the report of the authors it was fixed in the "cadaveric position") can in no way be explained by a *cortical* lesion.

Physiological and pathological experience has shown that in the cortex of man and of the higher mammals so far examined (except the cat), not the *respiratory* but only the *phonatory* function of the larynx is specially represented. Thus, even if according to Masini's view, an isolated cross-effect could be exercised from one *phonatory* centre, the *vocal* but not the *respiratory* function of the larynx would suffer, the latter being

mainly, if not exclusively, governed by the *medulla*. The *absolute* immobility of the vocal cord in Garel and Dor's case, therefore, cannot possibly be traced up to a cortical lesion. Our view was not merely theoretical, for with sufficient caution one can extirpate *the entire cerebrum, including the internal capsule* down to the floor of the fourth ventricle, without interference, after the first shock, with the *respiratory movements of the vocal cords*.

The reply of the French authors to these objections was unsatisfactory. They declared: 1. That the microscopic examination of the medulla in their case had been unnecessary ("nullement necessaire.") 2. That they had spoken of cerebral only but not of medullary conditions.

In our final reply we left the decision as to the former point to the readers of the controversy, and concerning the second laid stress upon the fact that in this question the cerebral conditions can not be discussed without ever keeping in sight the medullary centres and the different laryngeal functions. From this point the principle involved lends importance to the controversy; and in all future cases of unilateral paralysis of a vocal cord, believed to be due to a lesion of the cerebrum on the opposite side, we must insist upon a *thorough microscopical investigation of the whole neuro-muscular apparatus involved, from the medulla downwards*, so as to exclude the possibility of a lesion below the higher centres.

I have here to make a few general observations concerning the relationship between clinical and pathologico-anatomical observation, and experiments on animals, so far as they bear upon the different conditions obtaining in paralysis of the abductors and in central paralyses. For it may be objected to my arguments that it is inconsistent, after objecting to the validity of experimental evidence in the question of abductor paralysis, to admit it in my criticisms of clinical and anatomo-pathological data concerning paralyses of cerebral origin. But this objection can tell only upon those who are personally unacquainted with experimental work. It is necessary, therefore, that purely clinical observers should realize that there exists in these two questions an enormous difference in the relative value of experimental investigation.

When dealing with cortical paralyses of the larynx, we stand on the very ground of experimental investigation. The localisation of special cortical areas for the motor innervation of the larynx, must be defined by experiment, just as experiment has

defined the localisation of other cortical areas. The physiological discoveries so made have uniformly been corroborated by pathological experience. The larynx forms no exception to this rule and experimentation has added to our knowledge concerning its functions, without clashing with the teachings of clinical observation.

From the moment the question was raised as to the existence in *man* of a special bilateral cortical area for voluntary phonation, I have insisted upon the fact that even in typical cases of right sided hemiplegia with motor aphasia, there is no *aphonia*. In such cases no motor disturbances of the vocal cords can be seen; hence in man also there must exist a *bi*lateral representation of the organs of phonation. Here therefore experiment and pathological experience agree. Now the seat of the *left* phonatory area corresponds to the seat of the *speech* centre, as shown to be highly probable by Horsley and myself in our investigations on monkeys; hence, if a unilateral cortical lesion could produce paralysis of the opposite vocal cord, it would follow that palsy of the right vocal cord and some phonatory trouble must occur in lesions of this area, nearly *as frequently and regularly* as aphasia and right-sided hemiplegia. But how few cases of this character have as yet been reported, though attention has already been long drawn to this point! Again, most of the cases thus reported are founded upon clinical observation only during life, the possibility of multiple lesions at a lower level being not excluded; whilst the records of few autopsies that have been made, are as already mentioned all deficient on some point or other.

But experiment teaches us even more. It shows us, what could indeed have been expected, that great differences exist in the central representation of the larynx, with regard to its *phonatory*, and to its *respiratory* function. It teaches us, that the former is mainly represented in the cortex, the latter in the medulla oblongata. It has even shown us how in a highly organised animal we can remove not only one or both phonatory centres, not only an entire cerebral hemisphere, but the *entire cerebrum* down to the floor of the fourth ventricle, without the *respiratory* movements of the vocal cords being altered in their character. *Such* experiments entitle us, when confronted with a case in which on the strength of a more or less incomplete clinical or pathological examination, a unilateral cortical lesion is said to have produced complete paralysis of the opposite vocal cord, to be sceptical, and suspect some fallacy of observation.

The case, however, is very different if, as in the question of

abductor paralysis, experiments are merely intended to *imitate a pathological process*, usually of a *chronic* nature. That imitations hardly ever come up to the original is as true in medicine as it is in other sciences. But if *nature itself* offers us experiments under conditions the diversity of which no experiment on animals can imitate, and yet with always the same results, we may well disregard any attempted imitation of pathological processes bearing a merely superficial likeness to its natural prototype and disallow the validity of any generalisation derived from such a delusive instance.

I am gratified here to find myself in agreement with no less an authority than Prof. Virchow. He, who in 1881 (149) had so warmly and eloquently defended experimental methods against prejudice, found himself three years later obliged to protest against *over-estimating* them in questions like the present. He was speaking of the ossification of the bone marrow at the border of an amputation wound, a process in which it is "the violence of the changes produced by the experiment" which distinguishes experimental from pathological phenomena. "Those observers," he says, in his discourse on metaplasia (150), "who demand that every organic process must first be demonstrated by vivisection deceive themselves, as it appears to me, about the importance of the experiment in such cases. Pathological conditions occur which may be observed with the same regularity as can be secured by experimental ways; nay, which are even preferable to experiment. . . . ."

In further illustration of this thesis I may be allowed to give the outline of a case which I propose to publish fully on a future occasion.

Some years ago a woman, aged about 48, came to the Throat Department of St. Thomas's Hospital. Her only complaint was difficulty of swallowing, and she stated that she felt an obstacle about the level of top of the sternum. The patient looked sallow and cachectic. A bougie easily passed down the œsophagus. Though the symptoms did not point to any laryngeal affection, the left vocal cord was found to be fixed immovably in the *median position*, otherwise the larynx, as well as all other organs, were normal. The patient's age, the apparently normal state of all other organs, the absence of a syphilitic history, led one to think of an infiltrating carcinoma of the œsophagus, which had not yet obstructed the canal itself, but had already to some extent involved the left recurrent laryngeal nerve. Iodide of potassium was prescribed without any effect. A fortnight later there occurred ptosis of the left eyelid, then paralysis of the left rectus externus, followed shortly afterwards

by complete left ophthalmoplegia. Sensory troubles in the mucous membrane of the left cheek, left sided deafness, left sided paralysis of the facial nerve, made successively their appearance (154).

By this time of course we knew we had to deal not with a peripheral but with a central lesion on the left side, gradually involving one cerebral nerve after another. The patient was now admitted into the hospital, and subjected, under the care of Dr. Bristowe, to an energetic treatment by inunction, and large doses of iodide of potassium. Her condition within the next few months improved so much that, though not cured, she could leave the hospital. The left vocal cord remained all along immobile in the median position. Syphilitic pachymeningitis was diagnosed at the time. A few months later the patient, having become much worse, came back to the hospital; the paralyses had re-returned with greater intensity; the left vocal cord was now fixed in the *cadaveric* position, and the voice, which had been formerly quite normal, had become almost aphonic. Signs of incipient paralysis were found in some of the right cranial nerves. The patient was submitted to the same treatment, but without success. The paralytic phenomena on the right side progressed, and after a while included *diminished outward mobility of the right vocal cord*. This paresis steadily increased, but before the stage of complete right abductor-paralysis had been reached the patient died comatose.

The autopsy corroborated the diagnosis of syphilitic pachymeningitis.

All the affected cranial nerves were found to be embedded, near their points of emergence from the cranium, in a half-gelatinous, half-fibroid mass, which had partly destroyed, partly compressed them. The vago-accessory nerves on both sides were involved.

The importance of this case is obvious. Nature had showed herself a perfect experimenter. This *one instance* supplies a clue to some of the most difficult problems which have arisen in the course of the controversy about abductor paralysis and contracture: Here is to begin with the transition of an originally observed left-sided abductor paralysis into *complete recurrent* paralysis. Here we have, as if to exclude the idea of a contracture primarily of the laryngeal muscles gradually passing into paralysis, *multiple* affections in other cranial nerve regions, all of which, though affected by the *same* process, showed only *paralytic*, and no irritative, phenomena. And if the objection were still raised that the proof was not complete, and that the original median position of the left vocal cord might possibly be due to some *irritation*, Nature enabled us to dispose of this doubt by allowing us to witness a *gradual diminution of the outward*

mobility of the right vocal cord, which cannot possibly be interpreted as an irritative phenomenon!

I hope I may not be misunderstood, when I ask what experiment could have faithfully reproduced the conditions of such a case?

Many years' clinical and experimental investigation leave no doubt in my mind that in these questions experiment cannot lay down the law to clinical observation, nor clinical observation to experiment. I feel equally certain there is no incompatibility between them, that the one *completes the other*, and that wherever a contradiction seems to arise the conflict is only *apparent*. With sufficient patience and care it can be always traced to defective clinical or pathologico-anatomical observation, to faults in the experimental method or to an incorrect way of stating the problem.

The importance of the questions just discussed which are of general as much as of special interest, has to some extent dwarfed the other problems of laryngeal nerve physiology and pathology, which have arisen since the introduction of the laryngoscope. Quite a number of important additions to our knowledge, however, are to be enumerated.

Thus the old question as to the primary source of the motor innervation of the larynx has recently attained a new phase through the papers of Grossmann (152) and Grabower (153). According to the former the superior laryngeal nerve in rabbits takes its origin from the upper, the recurrent laryngeal from the middle bundle of the vagus. Grabower's experiments on animals also led him to consider the vagus and not the accessory as the motor nerve of the larynx. But Koch (154) on anatomical grounds supports the generally accepted view that the spinal accessory is the motor nerve of the larynx.

Hints (155), in experiments undertaken by the degeneration method on rabbits, finds that the spinal accessory receives, in addition to those, from the motor and spinal roots, fibres from the jugular ganglion of the vagus, and that the recurrent laryngeal consists mainly of accessory fibres. Dees (156) on the other hand shares Grabower's view that the accessory is not the motor nerve for the larynx.

This problem, which after Schech's paper (*l.c.*) appeared to have been definitely solved in favour of the view that the spinal accessory is the only motor nerve for the laryngeal muscles proper,

except the crico-thyroid, is thus again in anything but a settled condition.

Without committing myself to a final opinion, I may mention that after section performed by Professor Horsley of the internal branch of the spinal accessory in a dog, I have seen the vocal cord of the same side at once recede into the cadaveric position, whilst the autopsy showed the *absolute integrity* of the corresponding vagus; and thus the results of Grabower and others are at present not intelligible to me. I shall return to this point when discussing cases of unilateral paralysis of the tongue, palate and larynx.

The innervation and the mode of action of the crico-thyroid muscle, is now also the subject of much controversy. Led by careful experiments, Hooper (157) arrived at the same conclusion as Magendie, that contrarily to the accepted opinion, the contraction of this muscle moves the cricoid cartilage towards the thyroid. This view, which had already previously been expressed by Jelenffy and Schech (*l.c.*), is shared by Moura-Bourouillou (158), Desvernine (159), Kiesselbach (160), and v. Beregszaszy (161).

The papers by Simanowsky (162), Exner (163), Onodi (164), and Breisacher (165), which discuss the innervation of the crico-thyroid and other laryngeal muscles are so contradictory between themselves that it is not possible as yet to form a definite opinion amidst such discrepancies.

The observations of paralysis of the crico-thyroid made by Heymann (166), and Kiesselbach (167) ought to be mentioned here; as well as the experiments concerning movements of the vocal cords produced by direct stimulation of the motor laryngeal nerves in man. Such experiments have been made by Gerhardt (168), von Ziemssen (169), Rossbach (170), Pauly (171), and Kaplan (172). But these experiments have also yielded such different results that further investigations are necessary before such data can be utilized.

The statement made by Hale White (173), that the recurrent exercises a trophic function upon the thyroid gland, and contains many fibres not of cerebro-spinal origin, has not so far received corroboration.

According to Moura (174) the inter-arytænoid muscle acts in concert with the glottis-openers, and Jacobson (175) ascribes the same function to the thyreo-arytænoid muscle.

The interesting experiment made by Fraenkel and Gad (176) by applying cold to the recurrent laryngeal, when the outward

movements of the vocal cord, innervated by the frozen nerve, cease sooner than the adductor movements, has already been mentioned, when the question of abductor paralysis was discussed. Onodi's (177) more recent papers, in corroboration of the law of the greater vulnerability of the abductors, are as yet mere preliminary communications, and hardly prove his thesis.

We now come to pathology. With regard to the central affections of the motor laryngeal nerves, we must first mention the corresponding chapter in Gottstein's (178) text-book of diseases of the larynx, which adduces an excellent collection of illustrative cases. Krause (179), Löri (180), and Neumann (181), have also made valuable contributions towards the etiology and symptomatology of laryngeal affections in central nervous disorders.

With regard to affections of the cerebrum proper, Arthur Schnitzler (182), Hirt (183), Scheinmann (184), and Michael (185), considering that the so-called functional aphonia is in the light of recent physiological investigations a *cortical* affection have treated it by hypnotism and suggestion.

A paper has been devoted by Rosenbach (186) to "Functional Paralysis of Articulate Phonation," under which term he includes cases in which there is apparent absence of all innervation as well as those in which only quantitative changes of innervation are present. B. Fraenkel (187), and Bresgen (188), describe as "Mogiphonia," the professional weakness or premature fatigue of the voice, an affection which, from the description of its authors, may as well belong to the class of central as of peripheral neuroses. A similar remark applies to the "inability of whispering" or "apsithyria," an affection described by J. Solis-Cohen (189), and Peltesohn (190). The interesting cases reported by Cartaz (191) have already been mentioned in connection with the question of abductor paralysis. In these, as well as in Broadbent's case (192), lesions were present, situated, in my opinion, *above* the medulla oblongata, though they are described by their authors as belonging to the domain of bulbar paralysis. My view would well explain the initial paresis of the *ad*ductors in voluntary phonation.

Among *bulbar* affections the first place is held by the laryngeal paralyses of *tabes dorsalis*. It is remarkable that, whilst motor paralyses of the larynx have met since 1860 with such general attention, the literature of tabid laryngeal paralysis, which within the last few years has enormously increased, dates

only from the year 1881. It is true that Féréol, Martin, Jean, Boudin, Krishaber, and others had previously drawn attention to the so-called "laryngeal crises" or attacks of spasmodic cough, which in more severe cases pass into laryngeal spasms, choking attacks, convulsions, and loss of consciousness. But it was only in 1881 that Cherchevsky (193), who collected older cases and added some new ones from Charcot's clinic, stated that in some of these cases there had been actual laryngeal paralysis, and thus opened this new pathological chapter.

The neglect which has so long attended these paralyses, though strange, admits however of a very natural explanation. In a recent monograph on "The Laryngeal Troubles of Tabes Dorsalis," by Dr. H. Burger, (Leyden, E. I. Brill, 1891), it is shown that in the great majority of cases of tabes, in which laryngeal paralyses have been observed, the paralysis met with is of the nature of *abductor* paralysis. This, unless bilateral and fully developed, easily escapes observation, as mentioned by Schech in 1871, and emphasized by myself ten years afterwards, because *it leaves the voice wholly unimpaired*, whilst the unilateral form does not cause any *respiratory* symptoms, and the bilateral only, when much developed. And just in cases of tabes, in which the patients often are confined to bed, and do not exert themselves, even more advanced stages of bilateral abductor-paralysis often cause no appreciable dyspnœa. It is only since we have learned to appreciate the *diagnostic* value of this form of paralysis, and begun to *look* for it in suspicious cases, as we do for optic neuritis, that the number of cases of tabes in which laryngeal paralyses have been observed, has increased. We shall allude here only to a few of the contributions, the complete literature of the subject being given in Burger's paper.

My first case of bilateral abductor paralysis in tabes having apparently fallen into oblivion, I will mention it here because it is the *first* one of bilateral abductor paralysis in tabes, which has been authenticated by laryngoscopic examination, and because it is the very case which gave rise to the development of the law of the greater vulnerability of the abductors in progressive organic nerve-lesions. It is my own fault that it has been generally overlooked, because I did not realise at the time (1878) the connection between the laryngeal symptoms, then of two years' date, and the other incipient tabid symptoms. I only later arrived at a correct understanding of its etiological conditions. The reader who will take the trouble of following up the case in the different writings in which I have referred to it (194), or look it up in Burger's mono-

graph (*l.c.* p. 9), where it is fully given, may be compensated for his trouble by the exceptional interest it presents. One of its most remarkable features is the circumstance that in it, as in the well-known case observed later by Weil (195), the laryngeal symptoms preceded the usual symptoms of tabes by *fully two years*.

Important papers concerning paralytic laryngeal symptoms in tabes have also been contributed by M'Bride (196), Munschina (197), Oppenheim (198), Landgraf (199), Ross (200), Saundby (201), Kraus (202), Martius (203), Küssner (204), Felici (205), Aronsohn (206), Lucas Championnière (207), Dreyfuss (208), van Gieson (209), and Fano-Marina (210).

The two problems now before us, and which, I think, ought to be solved in the near future are:—(1) The relative frequency of these symptoms; and (2) the mutual relationship between crises and paralyses.

Concerning the first point apparently irreconcilable statements have been made by different authors. The explanation is, in my opinion, to be found in the fact that these statements are mostly based upon *too small* a material, and my own experience concerning this point is instructive enough. When I first began to study this question from the clinical point of view in the National Hospital for Paralysis and Epilepsy, I found among the first twelve cases of tabes which I examined not less than seven, in which either unilateral abductor paralysis, unilateral recurrent paralysis, or bilateral advanced paresis of the abductors, was present. I was surprised at this enormous proportion, and could hardly understand how, in these circumstances, the participation of the larynx in tabes should so long have escaped notice. On continuing, however, my observations, by methodical laryngoscopy of *every* case of tabes which came into the Hospital, I did not find among the next fifty to sixty patients *a single case* with paralytic laryngeal symptoms! In a more recent series of observations extending over fifteen months, and comprising all the in-patients suffering from tabes admitted into the Queen Square Hospital, I found in 27 cases of tabes seven instances of unilateral or bilateral paralysis or paresis of the glottis-openers. It is easy to understand from these statements how it happens that the reports on the frequency of laryngeal paralysis in tabes by authors who have worked on a comparatively small material only should be so widely discrepant.

Concerning the mutual relations of crises and paralysis no necessarily causal connection according to my experience, exists

between them. In a number of cases paralyses (unilateral or bilateral paresis or paralysis of the abductors or complete recurrent paralysis) are met with without any previous crises; in a second series, no paralysis ensues, even after occurrence of frequent and severe crises; whilst in a third series both, spastic and paralytic, phenomena are met with together in the same case. Should paralyses be produced, the law of the greater vulnerability of the abductors holds good. The spastic phenomena must be traced, I think to an increased irritability of the adductor centres. A peripheral stimulus conducted along the centripetal fibres of the superior laryngeal to those centres which are, according to this theory, in a condition of increased irritability, does not set up a mere cough, as under normal conditions, but spasmodic coughing, spasm of the glottis, general convulsions, in short a "crisis." This view also explains naturally the otherwise obscure influence of applications of cocaine upon the larynx, the benefit of which is mentioned by several authors (Gerhardt, Landgraf, Krause, &c.), the peripheral stimuli being temporarily cut off from the excitable centres. I shall conclude these remarks with a translation of the conclusions of Burger in his excellent monograph.

1. "The medulla oblongata is very frequently involved in the pathological process of tabes dorsalis.

2. Among bulbar nerves the vago-accessory is by far the most frequently attacked.

3. The morbid phenomena observed in the laryngeal nerves in tabes are motor paralyses, troubles of co-ördination, and so-called laryngeal crises.

4. The motor paralyses are the commonest form of laryngeal lesion; they often occur, however, without producing any symptoms.

5. The literature of tabid laryngeal paralyses furnishes a complete corroboration to Semon's law. Abductor paralysis is the tabid laryngeal paralysis "par excellence." No authenticated reports exist of isolated adductor paralyses.

6. The course of these paralyses is usually slow and progressive; the prognosis is always unfavourable.

7. In progressive tabid laryngeal paralysis, next to the posticus, the internal thyreo-arytænoid muscle is the adductor first affected.

8. Transitory functional adductor paralyses occurs occasionally in the progress of tabes.

9. Paralyses in the domain of the superior laryngeal are rare

in tabes, and have been observed only in connection with paralysis in the region of the inferior laryngeal nerve.

10. Other paralytic phenomena in the territory of the accessory nerve are pretty often associated with the laryngeal paralysis.

11. The tabid fixation of the vocal cords in the median line is a consequence of secondary contracture of the adductors, which has been superadded to a primary paralysis of the posticus.

12. Ataxy of the vocal cords exists.

13. In nomal abducting and adducting movements both the antagonistic groups of muscles of the larynx are probably innervated. Both physiological analogy (movements of the limbs) and pathological phenomena (*motus perversus* in respiration in cases of abductor paralysis and the tabic ataxy of the vocal cords) support this view.

[I have taken exception to this view in my paper on the position of the vocal cords in quiet respiration already quoted, and in my review of Burger's Monograph *Internationales Centralblatt für Laryngologie, &c.*, 1892, *p.* 354 *et seq.*]

14. Under the collective name of "laryngeal crises all attacks of dyspnœic cough observed in tabes have been put together. These are of a threefold nature: (*a*) Attacks which are due to defective closure, or anæsthesia of the aditus ad laryngem. (*b*) The laryngo-spastic attacks of suffocation which accompany abductor paralysis. (*c*) The laryngeal crises in the strict sense.

15. The laryngeal crises proper (*c*) occur either alone, or connected with laryngeal paralysis.

16. They are due to a condition of irritability in the sensory tracts of the laryngeal nerves, usually connected with an increased reflex excitability of the adductor centres.

17. The irritation which produces the attacks depends upon a chronic degeneration of the nerve tissue, and is usually localised in the *medulla oblongata*.

18. The anatomical basis of tabid laryngeal paralysis consists in a degeneration of the vago-accessory nuclei or fibres in the medulla, coupled with degenerative atrophy of the vagus and recurrentes, and of the paralysed muscles (*Mm. crico-arytænoidei postici*)."

Such are Burger's conclusions. This is not the place to enter more fully upon the interesting questions raised, and which form a fruitful field for further research.

Among other medullary affections we may mention the interesting association of unilateral paralysis of the tongue, palate and larynx, first described by Hughlings Jackson (211), and after

him by Stephen Mackenzie (212), Barlow (213) (no laryngoscopic examination), Oltuszewski (214), Limbeck (215), Turner (216) and (?) Jonathan Wright (217). The main interest of these cases was considered to lie in the light which this curious association throws upon the innervation of the *soft palate*, inasmuch as it implies that the motor innervation of this organ is supplied by the accessory, in agreement with the experimental results of Horsley and Beevor (218), and of Horsley and myself (219). These cases gain still further interest from Grabower's assertion (see above) that the pneumogastric, not the accessory, supplies motor innervation to the larynx. This assertion is the less supported by these cases, that in several the cucullaris and trapezius were also paralysed, and thus *both* branches of the accessory were affected, whilst there was no symptom pointing to an affection of the vagus.

Among other papers concerning laryngeal bulbar paralyses, I need only mention some well described cases by Eisenlohr (220); and Remak's (221) case of bilateral paralysis of the spinal accessory nerves. Not much can be made, I think, of Lenmalm's (222) case of paralysis of the vocal cords in amyotrophic lateral sclerosis.

Finally we come to *peripheral* affections, concerning which we find papers by Jankowski (223) and Rotter (224) (laryngeal paralysis after extirpation of goitre), Bäumler (225) (paralysis of the recurrent laryngeal nerve in chronic affections of the lungs), Newman (226) (laryngeal symptoms due to pressure of aneurisms upon the vagus and the recurrent laryngeal nerves), Möser (227) (laryngoscopic image in complete unilateral paralysis of the vagus nerve).

Comparatively little attention has been paid to the paralyses of the muscles antagonistic to the abductors, if we except the central cases, with functional paralysis of the entire adductor apparatus. Still, here also a number of interesting papers may be recorded. Thus Frank Donaldson, jun. (228), reports a case of paralysis of the lateral adductor, and W. R. H. Stewart (229) two cases of unilateral adductor paralysis due to reflex influences from the nose. French laryngologists have thoroughly studied the genesis, symtomatology and treatment of paralysis of the transverse arytænoid muscle, the papers of Lecointre (230), Lermoyez (231), and Proust and Tissier (232) deserving special mention.

I am at the end of my description, which, let me repeat, is not intended to refer to *every* publication made concerning the question of motor laryngeal paralyses, in the course of the last thirty years, but only to those which, according to my conviction, have contributed some solid elements to our knowledge. I have hardly alluded to Sensory paralyses and Spasmodic affections of the larynx.

Nevertheless, I venture to believe that those readers who have not paid special attention to the questions treated in this paper will, after reading it, be surprised to discover so great an activity in this apparently remote corner of medical investigation, and to find how the questions which move scientific laryngology overlap other domains of pathology. More has been done within the last thirty years on these topics alone than during the whole prelaryngoscopic period. But few of the questions raised, however, have been definitely settled, and on almost all the points to which I have had to refer, important problems still await elucidation. And, again, many points will become the objects of investigation in the near future, of which we have hitherto hardly had a glimpse.

I conclude with expressing the wish that in the further development of the subject we may be spared any further attempt at founding a special laryngeal pathology; and that it may be always remembered that the true scientific course is to refrain from premature explanations of observed facts. The true causation must eventually be reached by quietly and patiently following up and carefully comparing observations. He who is led away by mere ingenuous views, like the man who follows a Will-o'-the-Wisp, loses time and often lands in a morass.

BIBLIOGRAPHY.

(1) *Internationales Centralblatt für Laryngologie.* 1885, Bd. 2, p. 319.
(2) *Handbuch der speciellen Pathologie und Therapie.* 4. Bd., Kapitel I, p. 440, 1876.
(3) *Jahrgang* 1860, No. 11, 1861, No. 27.
(4) *Virchow's Archiv.* Bd. 27, S. 68, and 296, 1863.
(5) *Berliner klinische Wochenschrift,* 1869, p. 383.
(6) *New York Medical Journal,* Nov. 1880, p. 460.
(7) Vgl. z. B. "Ueber Diagnose und Behandlung der Stimmbandlähmung," *Volkmann's Sammlung klinischer Vorträge.* No. 36, 1872. "Laryngologische Beiträge." *Deutsches Archiv. für klin. Medicin,* 1873, Bd. 11, p. 575. "Stimmbandlähmung und Icterus," *Deutsche med. Woch.* No. 10, 1887, etc., etc.
(8) "Ueber Stimmbandlähmung." *Wiener medicinische Presse,* 1866, No. 417, ff.

(9) "Ueber Stimmbandlähmung." *Deutsches Archiv. für klin. Med.*, No. 2, p. 515, 1867, *and Transactions of the Pathological Society of London*, vol. 23, p. 66.
(10) *Trans. Path. Soc.*, vol. 24, p. 42.
(11) "Stimmbandlähmungen." *Deutsches Archiv. für klin. Med.*, Bd. 4, p. 376.
(12) "Cases of Laryngeal Paralysis." *Medical Times and Gazette*, 3. April, 1869.
(13) "Ueber Recurrenslähmungen." *Deutsches Archiv. für klin. Medicin*, Bd. 6, p. 37, 1869.
(14) "Cases of Aphonia from Paralysis of Instrinsic Muscles of the Larynx." *Amer. Journ. of Med. Science*, p. 305, 1870.
(15) "Diagnostique de la paralysie motrice des muscles du larynx." Paris, 1872.
(16) *Edinburgh Medical Journal*. July, 1881.
(17) *Transactions of the Pathol. Society*. 1882, vol. xxxiii., p. 82.
(18) *Sperimentale*. März, 1884.
(19) *Wiener Med. Presse*. No. 20, 1882.
(20) *Berliner klin. Wochenschrift*. No. 50, 1882.
(21) *Ibidem*. No. 1, 1883.
(22) *Klinik der Krankheiten des Kehlkopfes*. Wien, 1866.
(23) "Hoarseness, Loss of Voice and Stridulous Breathing in relation to Nerve and Muscle Affections of the Larynx." London, Churchill & Sons, 1868.
(24) "Diseases of the Throat and Nasal Passages." Second edition, Philadelphia, 1879.
(25) "Ueber die Functionen der Nerven und Muskeln des Kehlkopfs." *Habilitationsschrift*. Würzburg, 1873.
(26) "Laryngoscopie an Thieren." *Berliner Wochenschrift*, 3. October, 1873.
(27) *Medical Times & Gazette*, p. 638, 1866.
(28) "Hoarseness and Loss of Voice," etc., p. 34.
(29) *Berliner klinische Wochenschrift*, 1872, No. 20 und 21 und 1870, No. 7.
(30) *Volkmann's Sammlung klinischer Vorträge*. No. 95.
(31) *Ueber die Paralyse der Glottiserweiterer. Deutsches Arch. für klin. Med.* Bd. 13, 1874, p. 107 ff.
(32) *Loc. cit.*, p. 462 ff.
(33) "Paralyse der Musculi crico-arytaenoidei postic." *Berliner klin. Wochenschrift*, 1879, No. 33.
(34) *Loc. cit.*, p. 460.
(35) *Transactions of the Clinical Society of London*, vol. xi., p. 141 ff.
(36) *Loc. cit.*, p. 453.
(37) *Berliner klin. Wochenschrift*, 1883, No. 46.
(38) "Diseases of the Throat and Nose," vol. i., 1880.
(39) *Breslauer ärztliche Wochenschrift*, No. 2 u. 3, 1880.
(40) *Berliner klinische Wochenschrift*, No. 17 u. 22, 1884, No. 8 u. 10, 1888.
(41) Vol. ii., No. 3, Juli, 1881.
(42) *Loc. cit.*, p. 205.
(43) *Loc. cit.*, p. 220.
(44) *Loc. cit.*, p. 594.
(45) The reports of cases relating to this question and published during the first few years after the publication of my paper, have been fully quoted in my paper, "Ueber die Lähmung der einzelnen Fasergattungen des Nervus Laryngeus inferior (recurrens), *Berliner klin. Wochenschrift*, 1883, p. 46, *et seq.* A contribution by the late Professor Elsberg (*Philadelphia Medical Times*, July 30, 1881) is important. It shows that as the abductor fibres are more vulnerable than the abductors to organic pathological influences attacking the whole nerve trunk, so they *recover more slowly and rarely* from such lesions. I have lately had the opportunity of repeatedly corroborating this observation (see for instance *British Medical Journal*, Nov. 30, 1889, Meeting of the Medical Society of London).
(46) *Transactions of the International Medical Congress*, London, 1881, vol. iii., p. 214 ff.

(47) *Loc. cit.* p. 218.

(48) "On the Position of the Vocal Cords in Quiet Respiration in Man, and on the Reflex Tonus of their Abductor Muscles," *Proc. Royal Soc.*, vol. xlviii., 1890.

(49) *Comptes Rendus du 8 Congrès International*, vol. iv., p. 43 ff.

(50) *Berliner klinische Wochenschrift*, 1883, No. 46 ff.

(51) How negligently some authors have proceeded here appears, *e.g.*, from the fact that in an otherwise excellent recent text-book on "Affections of the Larynx," by I. E. Moure ("Leçons sur les Maladies du Larynx," 1890, p. 313 *et seq.*) the author represents me as the *originator* of the different hypotheses, which I have *contested* in my first paper, and devotes to me a long polemic!

(52) *Berliner klin. Wochenschrift*, 1884, p. 138.

(53) *Ibid.*, 1884, No. 14.

(54) *Ibid.*, 1884, p. 734.

(55) "Bericht über die Sommersaison 1884 im Schwefelbad Langenbrücken," Bruchsal, D. Weber, 1885.

(56) "A Contribution to the Study of Laryngeal Paralyses," *Edinburgh Med. Journal*, Juli, 1885.

(57) "Zwei Fälle von doppelseitiger Lähmung der Musc. crico-arytaenoidei postici," *St. Petersburger med. Wochenschrift*, No. 26-28, 1884.

(58) "Ophthalmoplegia, etc." BRAIN, October, 1885.

(59) "Aneurism of the Ascending and Transverse Aorta, etc." *British Med. Journal*, 19th December, 1885.

(60) "A Case of Paralysis of the Abductors of the Vocal Cords, etc." *St. Bartholomew's Hospital Reports*, vol. xxii., 1886.

(61) "Bilateral Paralysis of the Dilator Muscles of the Glottis with subsequent Paresis of the Constrictors." *Lancet*, 16 and 23 July, 1887.

(62) "Paralysis of the Abductors of the Vocal Bands." *Lancet*, 22 and 29 October, 1887.

(63) "Extirpation eines Cavernoms, etc." *Berl. klin. Woch.*, No. 7, 1888. "Traumatische Sympathicus-Hypoglossus- und Accessoriusparalyse." *Ibid.*; "Bemerkungen zu Remak's Fall, etc." *ibidem*, No. 8.

(64) "Zur Pathologie der Glottiserweiterung." *Deutsche med. Woch.* No. 26-28, 1888.

(65) "Ueber Recurrenslähmungen." *Münchener med. Wochensch.*, No. 51, 1888.

(66) "Ein Fall von linksseitiger Stimmbandlähmung, etc." *Charité-Annalen* xiii., 1888.

(67) "Zwei bemerkenswerthe Fälle von Stimmbandlähmung." *Ibid*, xiv., 1889.

(68) "Die Lähmungen der Kehlkopfmuskulatur im Verlaufe der Tabes dorsalis." *Virchow's Archiv.* 120, i., 1890.

(69) "Klinische Beiträge zur Kenntniss der Innervation, etc., des Kehlkopfes." *Orvosi Hetilap*, No. 25 ff., 1890, und *Pest. Med.-chir Presse* No. 36, 1890.

(70) *Berliner klin. Wochenschrift*, 1881, No. 50.

(71) *Archivi italiani di Laryngologia*, 1882, p. 52, und *Bolletino della Malattie dell' Orecchio*, No. 2, 1886.

(72) BRAIN, vol. v., p. 53, and "Diseases of the Nervous System," p. 199.

(73) BRAIN, vol. vi., p. 266.

(74) *Ibid*, vol. iv., No. 14 and 15.

(75) "Kehlkopfkrankheiten," *Erste Auflage*, 1884, Kapitel über Motilitätsneurosen.

(76) "La Paralisis de los Musculos Crico-aritenoides post, etc." Madrid, 1884. T. Amparo.

(77) "Etude sur la Contractilité Post-mortem, etc." *Arch. de physiol. normale et pathol.*, No. 6, 1885.

(78) "On an apparently Peripheral and Differential Action of Ether upon the Laryngeal Muscles." *Brit. Med. Journal*, 28th August and 4th September, 1886.

(79) "Roaring in Horses." London, Baillière, Tindall & Cox. 1889.

(80) "Roaring in Horses." *Illustrated Naval and Military Magazine*, vol. ix., August, 1888.

(81) "Ueber doppelseitige Recurrenslähmung." *Wiener medizinische Presse*, No. 18, 1882.

(82) "Some points in relation to the diagnostic significance of immobility of one vocal band, etc." *American Journal of Medical Sciences*, July, 1883.

(83) "Paralysis of the left vocal band in extreme abduction." *Transactions of the American Laryngological Association*, 1884.

(84) "Les fibres abductrices des Recurrents sont-elles toujours affectées les premières"? *Revue Mensuelle de la Laryngologie*, No. 5, 1886.

(85) *Berliner klinische Wochenschrift*, 1883, No. 46 ff.

(86) *Deutsche med. Wochenschrift*, No. 27, 1885.

(87) "Note sur la paralysie laryngée d'origine centrale." *La France Médicale*, No. 134 et 135, Vol. ii., 1885.

(88) *Berliner klinische Wochenschrift*, No. 35, 1884. (In cases of undoubtedly *bulbar* lead-paralysis of the laryngeal muscles the law of the greater vulnerability of the abductors is exemplified. (See for instance, Remak, "Saturnine Hemiatrophie of the Tongue." *Berliner klinische Wochenschrift*, No. 25, 1886.)

(89) I have demonstrated specimens of this kind before the Pathological Society of London and before the Laryngological Section of the Berlin Naturforscherversammlung in 1886.

(90) "Quelques reflexions à propos de l'affection dite paralysie des dilatateurs de la glotte." *Annales des maladies de l'oreille, etc.* Janvier, 1886. "Paralysie des dilatateurs de la glotte." *Ibid*, Septembre, 1886, etc.

(91) "Sur la paralysie des muscles dilatateurs de la glotte." *Bull. de la Soc. des Sciences méd. du Grand-Duché de Luxembourg*, 1885.

(92) "Etude anat. et pathol. des ganglions peritrachéo-laryngiens." *Annales des mal. de l'oreille, etc.* März, 1884.

(93) "Diagnosis of Diseases of the Brain," 1885, p. 101.

(94) "Endemic Goitre or Thyreocele." London, J. and A. Churchill, 1885.

(95) *Transactions of the Pathological Society of London*, 1882.

(96) "Innervatie. Functie en Verlaming van enkele Larynxspieren." Leiden, S. C. van Doesburgh, 1886.

(97) *Sitzungsbericht der k. k. Akademie der Wissenschaften in Wien*, vol. lxxxix. *Abth.* 3, 1. u. 2. Heft, 1884.

(98) "Studien über Innervation und Atrophie der Kehlkopfmuskeln." *Ibid*, vol. lxxxv.

(99) "Zur Anatomie der Kehlkopfnerven." *Ibid*, vol. lxxxv.

(100) See, *e.g.*, a case from the second table of my paper in the *Berliner klinische Wochenschrift*, 1883; also the paper above quoted by Percy Kidd; and my own case, which will be published in full in this paper, etc.

(101) "Three Cases of Pressure upon the Recurrent Laryngeal Nerve." *Transactions of the College of Physicians of Philadelphia*, May 5, 1886.

(102) "Beiträge zur Anatomie, Physiologie und Pathologie der Kehlkopfmuskeln." *Berliner klin. Wochenschr.*, No. 34 u. 36 und *New York Med. Jour.*, Aug. 31st, 1889.

(103) "Contribution à l'étude des sténoses glottiques chez les tuberculeux." *Annales des mal. de l'oreille, etc.*, Januar, 1887.

(104) "Medianstellung des Stimmbandes bei Recurrenslähmung," &c. *Virchow's Archiv.*, vol. 120, 124, 126.

(105) "Ueber physiologische Verschiedenheiten der Skeletmuskeln." *Breslauer ärztliche Zeitschrift*, No. 18 u. 24, 1883 und "Zur Anatomie und Physiologie der quergestreiften Muskeln." *Recueil Zoologique Tome, I.*, No. 4 1884.

(106) "Ein Versuch zur Erklärung des Unterschiedes im Verhalten der Glottisschliesser und -erweiterer bei Kehlkopflähmungen centralen Ursprungs" (in Russian), *Getchen klin. Gaz.*, No. 26 to 29, 1885. Referat im *Internationalen Centralblatt für Laryngologie, etc.*, Febr., 1886.

(107) "On an apparently peripheral and differential action of ether upon the laryngeal muscles." *B. M. J.*, 4 and 11 Sept., 1886.

(108) "The respiratory function of the human larynx." *New York Med. Journal*, July 4, 1885.

(109) "The function of the recurrent laryngeal nerve." *The Amer. Journ.*

of the Med. Sciences, July 1886, and *New York Med. Journal*, 6 and 13 Aug., 1887.

(110) *Brit. Med. Journ.*, 4 and 11 Sept., 1886.

(111) "Untersuchungen und Studien über Contracturen der Stimmbandmuskeln." *Virchow's Archiv.*, Bd. 98, Heft 2, p. 294.

(112) "Zur Aetiologie der Stimmbandlähmungen." *Deutsches Archiv für klin. Med.*, vol. 37, Heft 6, 1885.

(113) "Quelques reflections à propos de l'affection dite paralysie des dilatateurs de la glotte." *Annales des maladies de l'Oreille et du Larynx*, No. 1, 1886.

(114) "Three cases of pressure upon the recurrent laryngeal nerve, etc." *Trans. of the College of Physicians of Philadelphia*, 1886.

(115) "Leçons sur les maladies du larynx." 1890, Paris, p. 313 ff.

(116) "Contributions à l'étude des névrites primaires du tronc du recurrent." *Revue Mensuelle de la laryngologie, etc.*, No. 20, 1889.

(117) *Verhandlungsberichte*, Jahrgang, 1883 u. 1884, No. 17 u. 18.

(118) *Comptes Rendus du Congrès.* Vol. iv., Copenhagen, 1886, p. 43 ff.

(119) "Ueber das Verhalten der Schliesser und Oeffner der Glottis bei Affectionen ihrer Nervenbahnen." *Virchow's Archiv.*, Bd. 99, Heft 1, 1885.

(120) *Internationales Centralblatt für Laryngologie, etc.* Oct., 1885, p. 87.

(121) *a.* "Ueber die Adductoren-Contractur (vulgo Posticuslähmung) der Stimmbänder." *Virchow's Archiv.*, Bd. 102, 1885.

b. "Zur Pathogenese der Adductoren-Contractur der Stimmbänder." *Deutsches Archiv. für klin. Med.*, Bd 38, Heft 5 u. 6, 1886.

(122) "Posticus-Lähmung oder Adductoren-Contractur." *Sitzungsberichte der Würzburger Physicalisch-Medicinischen Gesellschaft*, 1885.

(123) "Aphonia und Dyspnoea spastica." *Wiener med. Presse*, No. 41 u. 42, 1885.

(124) "Lähmung der Glottiserweiterer als initiales Symptom der Tabes dorsalis." *Berliner klin. Wochenschr.*, No. 13, 1886.

(125) *Tageblatt der 59. deutschen Naturforscherversammlung.* Berlin, 1886, p. 226.

(126) The excerpts from Krause's and Remak's observations in the discussion have been taken from a shorthand report which was made for the "Internationales Centralblatt für Laryngologie" of the Meeting of the Laryngological Section of the Naturforscherversammlung, 1886. This report is in my possession and will be published.

(127) "Ueber die Beziehung des Kehlkopfs zum motorischen Nervensystem," *Deutsche med. Wochenschrift*, 1890, No. 31.

(128) *Transactions of the American Laryngological Association*, vol. 7.

(129) *Brit. Med. Journal*, 4 and 11 Sept., 1886.

(130) "Versuche über die Ausfallserscheinungen der Stimmbandbewegung bei Abkühlung des Nervus recurrens." *Centralblatt für Physiologie*, 11 Mai, 1889.

(131) "The Functions of the Brain," 1876.

(132) "Traumatismes cérébraux," 1878, p. 1842.

(133) "Ueber die Beziehungen der Gross-hirnrinde zu Kehlkopf und Rachen." *Sitzungsber. d. königl. preuss. Akad. d. Wissensch. zu Berlin*, Nov., 1883, und *Arch. f. Anat. u. Physiol. Phys. Abth.*, 1884.

(134) *Brit. Med. Journal*, 21 Dec., 1889; *Berliner klin. Wochenschrift*, 1890, No. 4. *Deutsche med. Wochenschrift*, 1890, No. 31. *Phil. Trans. of the Royal Society of London*, vol. clxxxi., B., 1890. This is not the place to enter more fully upon the unnecessary and unpleasant controversy which Krause began after the publication of our preliminary communication in the *British Medical Journal*, in defence of his priority, *which we had admitted on four occasions* in 1886 and 1887. This controversy will be found in the early numbers of the *Berliner klinische Wochenschrift* of 1890. I mentioned it here because the replies by Horsley and myself contain points of importance about laryngeal paralysis.

(135[a]) *British Medical Journal*, 1890.

(135[b]) "Note on Bilateral Degeneration in the Pyramidal Tracts, etc." *Brit. Med. Journal*, 4 January, 1890.

(136) "Leçons sur la fonction motrice du cerveau." Paris, 1887, p. 146 to 148.

(137) *Archivi italiani di Laryngologia.* Napoli, April, 1880, p. 45.

(138) "Zur Frage der Localisation des Kehlkopfs in der Grosshirnrinde." *Berl. klin. Wochenschrift*, No. 25, 1890.

(139) The following description is identical with that given by Horsley and myself in our paper "On the Central Motor Innervation of the Larynx" (*Transactions of the Royal Society*, 1890).

(140) The literature of these cases will be found in Masini's, Garel and Dor's, and Rossbach's papers, to be presently discussed. I do not refer to them individually, because the authors named themselves admit that none of them possess a decisive value for our purpose.

(141) *Comptes Rendus des travaux de la Section de la Laryngologie du huitième Congrès International periodique des Sciences médicales. Copenhague*, 1884, p. 70.

(142) *Transactions of the American Laryngological Association*, 1888, p. 195. Vgl. a. *New York Med. Journ.*, 22 June, 1889.

(143) "Localisation des corticalen Stimmcentrums beim Menschen." *Deutsches Archiv für klin Medizin*, Bd. 46, März, 1890.

(144) "Zur Pathologie der Motilitätsstörungen des Kehlkopfs, etc." *Allg. Wiener Med. Zeitung*, No. 22, 1890.

(145) *Annales des Maladies de l'oreille et du larynx*, April, 1890.

(146) *Ibid*, Mai and Juni, 1890.

(147) *Ibid*, Mai, 1886.

(148) *Lancet*, 1886.

(149) *Trans. of the 7th Med. Congress*, London. 1881, vol. i.

(150) *Vortrag, gehalten auf dem Internationalen Medicinischen Congress in Kopenhagen.* 1884, Separatabdruck, p. 14.

(151) I here report from memory, inasmuch as the notes of the case are at present in Dr. Bristowe's possession, and it is possible that the *order of occurrence* of the different paralyses may have been slightly different.

(152) *Wiener klin. Wochenschrift*, No. 49, 1889, No. 1, 3 u. 5, 1890.

(153) *Centralblatt für Physiologie*, No. 20, 1890.

(154) *Hospitals Tidende*, No. 20, 1890.

(155) *Pesther Med.-Chir Presse*, No. 23, 1888.

(156) *Archiv. für Psychiatrie*, No. 20, 1 Oct., 1888.

(157) *Transactions of the American Laryngological Association*, 1883.

(158) *Comptes Rendus de la Société française d'Otologie et de Laryngologie*, 1884.

(159) *Journal of Laryngology*, Feb. 1888.

(160) *Monatsschr. für Ohrenheilkunde*, No. 3, 1889.

(161) *Arch. f. d. gesammte Physiologie*, 46, 10, 1890.

(162) *Getschen. klin. Gaz.*, No. 26, 1887, und *Pflüger's Archiv.*, Bd. 42, 1888.

(163) "Bemerkungen über die Innervation des Musculus Crico-thyreoideus." *Arch. f. d. gesammte Physiologie*, Bd. 43, 1, 1888. "Ein physiologisches Paradoxon, betreffend die Innervation des Kehlkopfs." *Centralblatt für Physiologie*, 6, 1889.

(164) "Beiträge zur Lehre von der Innervation und den Lähmungen des Kehlkopfs." *Monatsschrift für Ohrenheilkunde*, No. 4, 1888.

(165) "Versuche über den Nervus Laryngeus superior." *Centralblatt für die med Wissenschaften*, No. 43, 1889.

(166) "Zwei Fälle von Lähmung des M. crico-thyreoideus." *Arch. f. klin. Med.*, vol. xliv., 5-6, Juni, 1889.

(167) "Zwei Fälle von Lähmung des M. crico-thyreoideus." *Arch. f. klin. Med.*, vol. xlv., 3-4, Oct., 1889.

(168) *Virchow's Archiv.*, 1863, vol. 27.

(169) "Die Electricität in der Medicin." Dritte Auflage, 1865, p. 198.

(170) *Monatsschrift für Ohrenheilkunde*, No. 10, 1881.

(171) Quoted by Kaplan.

(172) "Experimenteller Beitrag zur electrischen Erregbarkeit der Glottismusculatur." *Inaug. Diss.*, Berlin, 1884.

(173) *Brit. Med. Journal*, 22 August and 28 Nov., 1885.

(174) *Revue Mensuelle de la Laryngologie*, No. 12, October, 1886.
(175) "Zur Lehre vom Bau und der Function des Musculus thyreo-arytaenoideus beim Menschen." *Arch. für microscopische Anatomie*, Bd. xxix, 1887.
(176) *Centralblatt für Physiologie*, 11 Mai, 1889.
(177) "Eine neue experimentelle Methode im Gebiet der Kehlkopfmuskeln und Nerven." *Berliner klinische Wochenschrift*, No. 18, 1889, und *Pester Med.-Chir. Presse*, No. 16, 1889. "Experimentelle Untersuchungen zur Lehre von den Kehlkopflähmungen." *Centralblatt für die med. Wissenschaften*, No. 15 u. 16, 1889. "Zur Lehre von den Kehlkopflähmungen." *Ibid*, No. 40, 1889.
(178) "Die Krankheiten des Kehlkopfs." 3. Aufl., 1890, Leipzig u. Wien, F. Deuticke, p. 344 ff.
(179) "Ueber Functionsstörungen des Kehlkopfs, etc." *Centralblatt für Nervenheilkunde*, No. 23, 1885.
(180) "Die durch anderweitige Erkrankungen bedingten Veränderungen des Rachens, des Kehlkopfs und der Luftröhre." Stuttgart, 1885, F. Enke.
(181) "Lecture on Aphonia, etc." *Glasgow Med. Journal*, July—October, 1885.
(182) "Ueber functionelle Aphonie, etc." *Internat. klin. Rundschau*, No. 10-14, 1889.
(183) "Ueber die Bedeutung der Suggestionstherapie, etc." *Wiener Med. Woch.*, No. 27-30, 1890.
(184) "Die suggestive Tonmethode, etc." *Deutsche med. Wochenschr.*, No. 21, 1889.
(185) "Beiträge zur. hypnot. Therapie." *Deutsche Medizinal-Zeitung*, 1889.
(186) *Deutsche med. Wochenschrift*, No. 46, 1890.
(187) *Ibid.*, No. 7, 1887.
(188) *Ibid.*, No. 19, 1887.
(189) *The Med. and Surg. Reporter*, 1 May, 1875, and 10 and 17 November, 1883.
(190) *Berl. klin. Woch.*, No. 30, 1890. Peltesohn gives the entire literature of the question.
(191) "Des paralysies laryngés d'origine centrale." *France médicale*, No. 134 u. 135, 1885.
(192) "Bulbar paralysis." *Brit. Med. Journal*, 15 May, 1886. Meeting of the Harveian Society.
(193) "Contribution à l'étude des crises laryngées tabétiques." *Revue de médicine*, 1881, Bd. i., p. 541.
(194) *Transactions of the Clinical Society of London*, vol. xi., 1878, p. 141. *Ibid*, vol. xii., 1879, p. 184. *Archives of Laryngology*, 1881, vol. ii., p. 215, Case 21. *Transactions of the Clinical Society*, vol. xxii., 1889, p. 373.
(195) "Lähmung der Glottiserweiterer als initiales Symptom der Tabes dorsalis." *Berliner klinische Wochenschrift*, No. 13, 1886.
(196) *Edinburgh Medical Journal*, July, 1885.
(197) *Thèse de Paris*, Oct., 1885.
(198) *Berliner klin. Wochenschrift*, No. 49, 1885.
(199) *Berliner klin. Wochenschrift*, No. 38, 1886.
(200) "BRAIN," April, 1886.
(201) *Lancet*, 18 Sept., 1886, and *Birmingham Medical Review*, Dec., 1886.
(202) *Berliner klin. Wochenschrift*, No. 23, 1886.
(203) *Ibid.*, No. 8, 1887.
(204) *Ibid.* No., 20, 1887. These cases have also been described at length by Wegener in his Thesis. Berlin, 1887.
(205) *Archivi Italiani di Laryngologia*, Fas. 2, 1887.
(206) *Deutsche med. Wochenschrift*, No. 26 u. 28, 1888.
(207) *Journal de Médecine et de Chirurgie Pratiques*, Oct., 1888.
(208) *Virchow's Archiv.*, vol. cxx., 1, 1890.
(209) *New York Medical Record*, 21 Dec., 1889.
(210) *Ricistz Sperimentali di Phrenatria*, 1890, und *Archiv. für Psychiatrie* 1889, Bd. 21, S. 156.
(211) *London Hospital Reports*, vol., 1864, *et seq.*, see also *Lancet*, 10 April, 1886. *Sitzung der Harveian Society.*

(212) *Brit. Med. Journal*, 3 March, 1883; *Trans. Clin Soc. of London*, vol. xix., p. 317, 1886; *Brit. Med. Journ.*, 30 March, 1889, Meeting of the Clinical Society.
(213) *Clinical Society's Transactions*, vol. xxii., p. 322.
(214) *Medycyna*, No. 21, 1187.
(215) *Prager Med. Wochenschrift*, No. 16, 1889.
(216) *Brit. Med. Journal*, 14 December, 1889. Meeting of the Hunterian Society.
(217) *New York Med. Journal*, 28 Sept., 1889. In Wright's two cases the palate was not affected.
(218) *Proceedings of the Royal Society*, vol. 44, p. 272.
(219) See previous footnote.
(220) "Ueber centrale Kehlkopflähmung." *Deutsche med. Wochenschrift*, No. 21, 1886. "Zur Pathologie der centralen Kehlkopflähmungen." *Archiv. für Psychiatrie*, vol. xix., 1888.
(221) *Deutsche med. Wochenschrift*, No. 27, 1885.
(222) *Upsala Läkareförenings Förhandlingar*, xxii., No. 7. 200. See also *Intern. Centralbl. f. Laryngologie, etc.*, Bd. iv., p. 413.
(223) *Deutsche Zeitschrift für Chirurgie*, Bd. xxii., 1885.
(224) "Die operative Behandlung des Kropfes." *Archiv. für klin. Chirurgie*, Bd. xxxi., 1884.
(225) *Deutsches Archiv. für klin. Medicin.*, vol. 37, Heft 3 u. 4, 1885.
(226) *Brit. Med. Journal*, 2 July, 1887.
(227) Mittheilungen aus der med. Klinik zu Würzburg, Bergmann, Wiesbaden, 1885.
(228) "Paralysis of the lateral adductor muscle, etc." *New York Med. Journal*, 12 February, 1887.
(229) "Two cases of unilateral adductor paralysis, etc." *Lancet*, 13 Oct., 1888.
(230) "Étude sur la paralysie isolée du muscle ary-aryténoidien." Bordeaux, 1888.
(231) *Annales des Maladies de l'Oreille, etc.*, Juli, 1885.
(232) *Ibidem*, Mai, 1890.

ON HYSTERIA.

BY J. MICHELL CLARKE, M.A., M.B., M.R.C.P.

(1) Leçons Cliniques sur l'Hystérie et l'Hypnotisme faites à l'Hôpital de St. André de Bordeaux par A. Pitres, Professeur et Doyen de la Faculté de Médicine de Bordeaux, &c. D'une Lettre-préface de M. le Professeur J. M. Charcot. Vol. i. and part of Vol. ii. Paris: Octave Doin, 1891.

(2) Traité Clinique et thérapeutique de l'Hystérie d'ápres l'enseignement de la Saltpêtrière par le Dr. Gilles de la Tourette, ancien chef de clinique des maladies du système nerveux à la Saltpêtrière. Préface de M. le Prof. J. M. Charcot. Vol. i., Hystérie normale ou interparoxystique. Paris: E. Plon Nourrit et Cie, 1891.

(3) Les Agents provocateurs de l'Hystérie par Georges Guinon. Paris: Bureau de Progrès Mèdical, 1889.

(4) L'Hystérie Male dans le service de M. le Prof. Pitres à l'Hôpital Saint André de Bordeaux par le Dr. Emile Bitot. Paris: 1890.

(5) Les Anesthésies Hystériques des Merqueuses et des Organes de sens et les zones hystérogènes des muqueuses par L. Lichtivitz. Paris: 1887.

(6) Des Anesthésies Hystériques par le Prof. A. Pitres. Bordeaux: 1887.

(7) Des Zones Hystérogènes et Hypnogènes par le Prof. A. Pitres. Bordeaux: 1885.

(8) Étude Clinique sur les troubles de la Sensibilité Cutanée chez les Alcooliques par le Dr. Edmond Grasset. Bordeaux: 1887.

(9) Quelques cas d'Hystérie Male et de Neurasthénie par le Prof. Grasset. Montpellier et Paris: 1892.

(10) Recherches Cliniques sur les troubles de la Sensibilité Cutanée dans la Chlorose par le Dr. P. G. Laporte. Bordeaux: 1888.

(11) Recherches Cliniques et thérapeutiques sur l'Épilepsie, l'Hystérie et l'Idiotie par Bourneville, médecin de Bicêtre. Paris: 1890.

(12) Comparaison des effects de divers traitements dans l'Hystérie précédée d'une esquisse historique sur la Metallothérapie par Oscar Jennings. Paris: 1878.

THE authors of the first three books have all been pupils of Prof. Charcot, and indirectly or directly, the works are the outcome of that systematic study of hysteria, initiated and carried on by him at the Saltpêtrière, which has been productive of such great results in extending our knowledge of the neurosis, and in

putting it on an accurate scientific basis. Both Prof. Pitres' and M. Gilles de la Tourette's books are preceded by a preface written by Prof. Charcot. The direct object of the latter work is indeed to bring together, in an easily accessible form, the substance of M. Charcot's teaching on hysteria. This first volume treats only of "normal" hysteria, that is to say, the ordinary state of the hysterical in the interparoxysmal periods; in it, only the common basis or foundation of the neurosis, as evidenced in the presence of permanent "stigmata" is considered, and not the convulsive seizures or other paroxysmal manifestations, which are to be dealt with in a second volume.

The work will thus form a systematic treatise on hysteria. It is written with perspicacity and clearness, and with a completeness that shows the author's extensive knowledge of his subject; nor must we forget to mention that his own original contributions to the study of hysteria have been numerous and important. The reader will find full information on every branch of the subject considered in this volume; and the full consideration of, and abundant references to, the work and views of other observers, both French and foreign, enhance its value as a book of reference. Perhaps we may specially mention the sections on disorders of vision, hysterical tremor, the mental state in hysteria, and the interesting historical notices which occur throughout the work.

Prof. Pitres has arranged his book on a somewhat different plan. It is based on the study of one hundred cases of hysteria taken from his clinical wards. In it will be found an admirably written account of the chief manifestations of hysteria, and one of the leading features of the book is the graphic way in which cases are brought forward—presented before the reader's eye, so to speak—in order to illustrate the subject under discussion. Such a mode of treatment is a very valuable aid to the memory in helping one to get a distinct mental picture of symptoms or groups of symptoms. It is impossible, in the following digest, to do justice to this character of the book, and to the life-like manner in which the clinical descriptions are given—a branch of the medical art in which, as is well-known, the French particularly excel. There are some interesting plates at the end of vol. i., representing the cures of various (hysterical) disorders that took place in the eighteenth century, at the tomb of the Deacon Pâris. Vol. ii., which is almost entirely occupied by the subject of hypnotism, was reviewed by Dr. Lloyd Tuckey in the last number of this journal.

The scope of M. Guinon's work is more limited than that of the other two, it has already been reviewed in BRAIN, vol. xii., p. 407, by Prof. Marie.

The following historical sketch is taken from M. Gilles de la Tourette's work. Plato first formulated the theory, which so long regulated the treatment and clinical study of hysteria; he thought that the uterus was an animal which ardently desired to engender children, and when after puberty it remained a long time sterile, it ran over the body, obstructed the entrance of air, stopped respiration, threw the body into extreme dangers, and caused divers diseases.

Hippocrates who was thoroughly familiar with epilepsy, does not appear in his work to always differentiate it very clearly from hysteria, with which he was certainly acquainted but much more imperfectly.

A passage from Celsus (1) is quoted in which this author clearly brings out the elements of the differential diagnosis between epilepsy and hysteria.

Hysteria flourished in the ignorance and religious fanaticism of the middle ages. Our knowlege of hysteria at this epoch is due to the labours of Charcot et Richet, who have in their works, "Les démoniaques dans l'art" (1887), "Les malades et les difformes dans l'art" (1889) and "Études Cliniques sur la grande hystérie ou hystéro-épilepsie," brought together a number of documents and pictorial representations of hysterical persons. One of the oldest is a miniature on the manuscript of the Emperor Otto preserved in the Cathedral at Aix-la-Chapelle, which represents the young man possessed with devils being brought before Christ; the patient is represented in the midst of an hysterical convulsion with the trunk forcibly bent backwards. A bas-relief in bronze from the gate of a church at Verona, represents a woman possessed with a devil; under the benediction of the bishop to whom she has been brought, the devil is represented, as going out of her mouth: the woman herself is bent backwards in form of the "arc de cercle" with a prominent and tympanitic abdomen. Both these examples date from the 11th century. There is a still earlier (5th century) representation of an exorcism in which the person possessed is in an hysterical attitude. Dr. de la Tourette notes that the possessed, *i.e.*, the hysterical persons were in two of these instances males.

The extensive epidemics of chorea of the middle ages were not true chorea, but rhythmical hysterical chorea—witness the representations by Breughel in 1574 of the "Dancers of St. Guy,"

which M. G. de la Tourette reproduces from "Les Démoniaques dans l'Art."

In 1618 Charles Lepois of Pont-à-Mousson anticipated many of the modern views upon hysteria. He recognized the existence of hysteria in children and in men; he gave a description of the sensory troubles premonitory of an attack, *i.e.*, obnubilation of sight and of hearing, loss of voice, and of the salivation following the attack; he recognized the paralysis of the upper and lower extremities. Sydenham's observations on the neurosis were the best and most complete up to the present century. The accuracy of his work was, however, not understood by his contemporaries, nor appreciated at its true value until the labours of Briquet in 1859. "With the eye of genius Sydenham had seen so many things which appeared so singular and incomprehensible at their first description, that he remained entirely misconceived." With the exception of Sydenham the authors of the 18th and of the first half of the 19th century lost themselves in the maze of empty and fruitless discussions which had a disastrous result on the evolution of the scientific knowledge of hysteria.

The work of Louyer Villermay (2) in 1816, denied the existence of male hysteria, and stated that the most frequent causes of hysteria were the deprivation of the pleasures of love, the chagrins arising from this passion, and menstrual irregularities. His views, as Briquet said, belonged to 1500 rather than to the year 1816. They were ably combated by Georget. The French writers about 1846, Brachet (3), Landouzy (4), do not seem to have been acquainted with the masterly lectures of Brodie (5) written 10 years previously. Brodie admits male hysteria, and in describing hysterical coxalgia says: "It is not the muscles which do not obey the will but the will itself which does not enter into action." He was the first to give the proper method of treatment for hysterical paralysis and contractions.

In 1859 appeared "Briquet's Treatise on Hysteria," which contains an immense amount of accurate clinical observation on hysteria. He concludes that the disease ought to be considered as a functional affection (*affection dynamique*). The various phenomena of hysteria are very exactly treated, but the general laws on which the complex symptoms depend are not mentioned, and their discovery, which has so revolutionised the knowledge of hysteria, dates from the labours of Professor Charcot at the Salpêtrière. Previously the differences between hysteria and epilepsy were ill defined, and it was thought that hysteria and epilepsy could be combined in the same patient as hystero-

epilepsy. Charcot showed that they are never combined, though they may exist separately in the same patient. Hystero-epilepsy contrary to appearances, is nothing but pure hysteria in its most developed form—hysteria-major. He retained the term hystero-epilepsy, applying to it this new meaning. In 1873 he described the hysterical convulsion as following certain laws which were enunciated for the first time. He also pointed out the existence of the "status hystericus" (état de mal hystérique) as opposed to the "status epilepticus" previously known. He described hysteria as a disease "one and indivisible" and gave clear indications for its diagnosis from epilepsy. To him we owe also the study of the permanent "stigmata" of the neurosis, the presentation of hysteria in the male from an entirely new point of view, and the working-out of the chemical phenomena of hysteria and of traumatic hysteria.

M. Pitres observes that in the middle ages the absorbing character of religious ideas, the frequent pictorial representations of demons, the burnings of persecution determined the form of hysteria; the patients were "possessed of devils." In our days the environment is changed, the dominant ideas are of another kind, and belong to the side of the affections, the affections, *e.g.*, contrarieties in love are more apt to produce hysteria. There has been no change in type of the neurosis; the "possessed of devils" and the patients of to-day both show the same disorders of sensation, contractions, paralyses and convulsions. Even now epidemics of "demoniacal" hysteria occasionally develop in remote districts and are very instructive. The epidemic of Morzine, a remote mountain village in the Haute-Savoie, where the people are illiterate and very superstitious, is a good instance. In 1857 a young girl saw her companion taken out of a stream half drowned, she fell down without consciousness, "as if dead." A few days later a friend who was with her in one of these attacks was similarly affected. The two girls afterwards had convulsive seizures, followed by periods of hallucination, in which they blasphemed horribly, uttered prophecies—some of which were verified—and climbed trees like monkeys. In the course of 8 months, 18 people were affected. Three patients were cured by their fathers threatening them with severe punishment if they continued to be convulsed. This being the state of affairs the ecclesiastical authorities held public exorcisms. The malady now rapidly spread and in 1861 about 120 people had had convulsive attacks. Some persons who were sceptical as to the alleged demoniacal origin of the fits were denounced as heretics, and one

barely escaped with his life from the popular fury. The epidemic was finally stopped by the government sending a force of gend'-armes, removing the parish priest, isolating the patients, and removing the worst cases to distant hospitals. It is unnecessary to point out the many analogies between this modern outbreak and those that are on record as occurring in the middle ages.

M. Pitres begins his work by enumerating five propositions which sum up the characters common to the manifestations of hysteria and serve as a basis for a clinical definition of the disease as a substitute for the nosological definition which is at present impossible.

(1) Hysterical phenomena result from purely functional disturbances of the nervous system.

(2) They may be abruptly excited, modified, or suppressed by psychical influences or physical agencies which have no effect on similar phenomena due to organic disease.

(3) They very seldom exist alone; in the immense majority of cases certain latent stigmata coexist with the conspicuous manifestations of the neurosis.

(4) They have no regular evolution; they come on in no pre-established order and proceed under different forms and at different times in the same subject.

(5) As a rule they have not that profound effect on the general health and mental state of the persons affected by them that similar phenomena dependant on another cause would have. The author lays especial stress as of real diagnostic value on the indifference and serenity of hysterical women in the presence of the various illnesses from which they may be suffering. They never believe themselves seriously ill and they never lose their self-consciousness, their regard for their appearance, &c.

The records of former centuries especially from the XIIIth. to XVIIth., when hysteria often took the form of epidemics prove that it is not a disease caused by our high state of civilisation.

The primary cause of hysteria, M. Charcot teaches, is heredity; this may be similar as when mother and daughter both suffer from the neurosis, or it may act by transformation, as in cases where the progenitors of the patient have suffered from some nervous disease other than hysteria. Apart from heredity, there exist only the "exciting" causes of the disease.

Briquet (6) found in 351 hysterical patients, whose families comprise a total of 1,103 people, 430 men and 673 women, that amongst their antecedent and collateral relatives 214 were hysterical, 13 epileptic, 16 insane, 1 had delirium tremens, 1 was

paraplegic, 3 somnambulists, 14 affected with convulsions, and 10 with apoplexy. Twenty-five per cent., therefore, of these 1,103 people suffered from affections of the nervous system. Persons born of hysterical parents are twelve times more predisposed to hysteria than those whose parents are non-hysterical. Sometimes hysteria, from the number of patients affected in one family, takes almost the character of a "family" disease. Hammond (7) states, that in 209 cases of hysteria where he had enquired into the question of heredity, 131 patients had mothers, grandmothers, or aunts affected with this malady, and in many others, the parents suffered from other nervous diseases. It is difficult to reconcile this statement with that of Russell Reynolds: (8) "Hereditary taint has not been shown to exert any marked influence in the development of hysteria." As regards male hysteria, Batault, (9) in his inaugural thesis compiled in the clinic of M. Charcot states, that of 100 cases of hysteria in men, a pathological heredity was traced in 77, and did not appear in the 23 others. The above 77 cases came from 75 families, there being two instances where two brothers were affected. In 56, one or other of the parents, or both, were affected with some nervous taint. The mother alone was affected in 35 cases, both parents in 12, and in 9 the father only. Thus, in both male and female patients, it is the mother who is the most often nervous.

Bodenstein, (10) in 11,225 cases of nervous disease in the clinic of Profs. Mendel and Eulenberg, found 1,224 hysterics, of whom 122 were men. He says "Heredity is shown still more often in men than in women." Bitot, (11) from the clinic of Prof. Pitres at Bordeaux, in 22 cases of male hysteria, traced a neuropathic heredity in 17, in 2 others the fathers were intemperate.

The influence of heredity, here direct heredity, is above all, marked in children; in 58 of 80 cases, varying from 1 to 12 years of age, the parents were hysterical, 2 were insane and 3 epileptic. (Briquet.)

Are such morbid agencies as syphilis and alcohol to be considered as exciting or primary agents; and are "arthritism" and tuberculosis in the parents capable of engendering hysteria in the offspring? M. de la Tourette thinks that arthritism, alcoholism, and syphilis in the parents only act by bringing out in the children the hereditary nervous taint which remains latent in the parents; and that outside the neuropathic group of diseases hysteria has no connections. At the same time, M. Charcot has shown that the uric acid diathesis, arthritism, syphilis, and tuberculosis, are often associated with the neuro-

pathic diathesis. "A neuropathic hereditary tendency is thus the primary cause of hysteria; the neurosis is a branch of the great neuropathic family. Its connections with arthritism, for example, are only those of contact and not of fusion. Hysteria is one in its heredity as it remains indivisible in its evolution."

In 272 cases of hysteria in children the onset occurred:

	GIRLS.	BOYS.	TOTAL.
Below 3 years in	19	1	20
At the age of 3 years	—	1	1
,, ,, 4 ,,	1	1	2
,, ,, 5 ,,	4	2	6
,, ,, 6 ,,	3	2	5
,, ,, 7 ,,	15	4	19
,, ,, 8 ,,	16	6	22
,, ,, 9 ,,	15	7	22
,, ,, 10 ,,	18	15	33
,, ,, 11 ,,	24	17	41
,, ,, 12 ,,	22	13	35
,, ,, 13 ,,	27	16	43
,, ,, 14 ,,	12	8	20
,, ,, 15 ,,	—	3	3
	176	96	272

The proportion of girls to boys is a little less than 2 to 1.

The influence of the appearance of menstruation in girls thus appears to be nil.

Women.—From statistics based on his own observations and on those of preceding authors, Briquet concluded (i.) one-fifth of cases of hysteria in women develop (in France) before the age of puberty; (ii.) rather more than a third of the cases develop from 15—20 years; (iii.) the frequency of hysteria rapidly decreases from 20—25 years of age, being only half as frequent as in the antecedent period. From 25—40 it remains at the same proportion, being ten times less frequent than from 12—20 years of age; (iv.) hysteria is very rare between 40 and 60 years and is not more frequent at any one year than at another during this period.

Men.—M. Batault shows that the date of the first appearance of symptoms in men is most often between the ages of 10 and 20 years. Of 192 cases of male hysteria in 44 cases the onset was between 10 and 15, and in 34 cases between 15 and 20. The predisposition due to age conforms much less to the period of

development of the genital organs than is generally supposed. M. Gilles de la Tourette thinks, judging from his experience at the Saltpêtrière, that the time of onset is later in men than in women, which is accounted for by the greater incidence of exciting causes, such as traumatisms and intoxications, during adult age in men. In 22 cases of hysteria in men reported by M. Bitot (12) the mean age of onset was 26 years.

What is the real frequency of hysteria? Sydenham wrote that as "fevers with their attendants constitute two-thirds of the diseases to which mankind are liable, upon comparing them with the whole tribe of chronic distempers, so hysteric disorders or at least such as are so called make up half the remaining third part, that is they constitute one moiety of chronic distempers."

With regard to the frequency of hysteria in man and its proportion to the number of cases in women, M. Bodenstein in 11,225 cases of nervous disease observed 1,224 hysterics of whom 122 were men or 1 to 10 women. This is double the scale of Briquet, who said that the proportion was 1 to 20.

In 100 cases of hysteria Pitres found 31 men and 69 women, or about 1 to 3.

M. P. Marie (13) while at the "Bureau central d'Admission," where patients make application for admission to hospital, examined during May, 1889, 697 persons affected with various diseases of whom 172 were women and 525 men. He sought to determine the frequency of hysteria in the two sexes, and to that end divided the patients who presented symptoms of hysteria into two classes (i.) "hystérie massive," those in whom there was well marked anæsthesia, general, unilateral, or limited to a limb, and ii. "hystérie mitigée," in which symptoms of hysteria were certainly present but not accompanied by disturbances of cutaneous sensation. Of the 525 men, 251 presented the characters of "hystérie massive," 3 of "hystérie mitigée," of the 179 women one only fell in the first, five in the second category. The percentage of cases in the two sexes of "hystérie massive" is therefore 4.76 per cent. men, 0.58 per cent. women. The male patients belong to the working classes, or those who have descended in the social scale and have therefore very frequently been exposed to the exciting causes of male hysteria, traumatisms, poisoning by alcohol, lead, &c. The social status of the women is a little higher and they are therefore less exposed to the action of the above causes. M. Maires thinks that the explanation of this very unexpected proportion between the two sexes lies in the fact that male hysteria is much more rare in the middle and upper classes

as compared with the lower, whereas this is not the case in hysteria in women. As regards the second class "hystérie mitigée," there is 1 case in 35 women and 1 in 175 men: this form is therefore much more frequent in women, and this is a fresh confirmation of M. Charcot's teaching that hysteria in men is generally characterised by its obstinacy and severity.

M. Souques (14) compiled the statistics of 441 men and 240 women under the care of M. Chauffard at the Broussais hospital and found amongst them 26 cases of hysteria amongst the men and 6 cases only amongst the women.

M. Gilles de la Tourette gives similar statistics derived from the records of the Salpêtrière, where the patients come from all classes and from all countries. During the two years from November, 1887 to November, 1889, there were, in the wards under his care as "chef de clinique," 323 patients, comprising 200 women and 123 men, of whom 67 women and 37 men were suffering from hysteria, in the proportion of 2 to 1. In the out-patient department of 726 patients of both sexes, there were 51 female and 21 male cases of hysteria or again about 2 to 1. M. Parinaud, the ophthalmic surgeon to the hospital, examined 49 women and 30 men affected by visual disturbances due to hysteria, but this great proportion of men is partly accounted for by the fact that many of the hysterical women, in whom the symptoms were well marked, were not sent to him for ophthalmoscopic examination, whilst all or nearly all the men were examined, and that a large number of cases of male hysteria were especially sent to M. Charcot from the different hospitals, on account of the interest he took in them. But even allowing for this, the number shows the frequency of male hysteria. (15) At the Salpêtrière hospital, therefore, where all social grades are represented, one case of hysteria in man occurs for every two or three in women.

M. Pitres attaches considerable importance, from the point of view of prognosis and prophylaxis, to the neuropathic symptoms shown in early childhood by those who are liable to become hysterical. Such are cataleptic attacks, suffocative attacks, certain varieties of cough, which resemble whooping-cough and last for weeks or months; attacks of hiccough or vomiting without sufficient cause, palpitation, night-terrors, headache during adolescence; certain forms of pulmonary congestion, of hæmorrhage from the nose, stomach, or lungs; somnambulism, contractions of the limbs, or temporary paralysis of motion or sensation, neuralgias, and pains in the joints.

Exciting causes of Hysteria:—

(i.) Strong moral emotions. Instances of emotions as exciting causes of hysteria abound. Fear is the most potent. Sometimes a single moral shock suffices to evoke hysteria, often it only appears after a longer or shorter series of repeated emotions. From Brodie to the present time all authors are in accord as the evil influence in this direction of an injudicious bringing up. M. Guinon instances first corporal punishment, or physical ill-treatment. Blows may act with double force provoking symptoms of hysteria by moral shock, and by traumatism. Religious practices carried to extremes are often the starting point of hysteria.

Equally effectual are the pernicious practices of deterring children from doing wrong by threatening them with a bogie or a policeman, and of allowing them to be constantly reading stories in which sorcerers and ghosts play prominent parts. M. Charcot (16) relates the very instructive history of an epidemic of hysteria which took place in a family much given to spiritualistic séances. The obvious lesson is to carefully protect children who are nervous and impressionable from all such influences.

(ii.) Alongside of the moral emotions we must place imitation as an exciting cause, the influence of which is well known in epidemics of hysteria. M. Charcot considers that the form of hysteria which is developed in a patient by imitation of the symptoms seen in other hysterics is not a very grave one, and is fraught with less evil to the future welfare of the patient than other forms of hysteria.

Attempts at hypnosis constitute one of the most effectual agents in the causation of hysteria. The convulsive attack is commonly the symptom which follows on injudicious hypnotisation. M. Guinon narrates a case in which the only hysterical manifestations were slight cough and hiccough, and in which extremely violent and frequent hystero-epileptic convulsions followed repeated unsuccessful attempts at hypnosis. He thinks that hypnosis should never be resorted to except in cases where the neurosis is present in so severe a form that there is no risk of making the patient worse.

Influence of traumatisms on the development of hysterical symptoms.—M. Guinon observes on this subject, that the name of "nervous shock" is applied to that state in which a person falls who has been the victim of a traumatism or of a more or less violent physical shaking, accompanied as it always is by strong emotion. It is a state in which the symptoms are partly physical,

partly psychical. By traumatic hysteria, we mean hysteria developed under the influence of an injury, and the accompanying nervous shock. Nervous differs from what is called traumatic "shock" (shock following severe operations and injuries). The latter is always a grave accident and often fatal, while the "former does not present any gravity in itself, at least in so far as regards the life of the patient."

The knowledge that such symptoms follows an injury dates from Erichsen's work. (17) The cause of the symptoms was considered by him to be an inflammation of the spinal cord and its membranes.

In 1882 appeared the first edition, the second followed in 1885, of Page's work on "Injuries of the spine and spinal cord and Nervous Shock:" in this book the brain was considered to be the primary seat of the functional troubles observed, and the term "Railway-Brain" was substituted for "Railway-Spine," the principal factors of the condition being neurasthenia and *hysteria*. The next step forwards came from America when Mr. Walton, (18) returning from the study of nervous diseases at the Saltpêtrière, attributed the various nervous symptoms that follow on railway and other accidents to hysteria.

In Germany the question took another aspect. Oppenheim and Thomsen (19) from the study of the symptoms following accidents, wished to establish a special neurosis, the traumatic neurosis, from the presence of such symptoms as hemianæsthesia, sensory anæsthesias, contraction of the visual fields, &c., from the obstinacy of the hemianæsthesia, as opposed to its variability in hysteria, and from the mental state of the patients. M. Charcot (20) however showed that hysteria is often provoked by a traumatism, and that the majority of the nervous symptoms recognised in the injured are in reality only hysterical symptoms identical with those present in hysteria from whatever cause it may arise.

The hypothesis of a distinct traumatic neurosis was further brought forward in a modified form by MM. Oppenheim, Strümpell and others, but the question seems now to be finally settled that the symptoms are due to hysteria, "traumatic hysteria;" this result is largely due to the labours of Charcot and his pupils. The author thinks that the cause of the confusion is, in great part, to be attributed to the prevailing ignorance of the symptoms of male hysteria.

M. Pitres observes that the effects of traumatisms differ according to the morbid predispositions of the subjects; in one

exciting an epileptic discharge, in another neurasthenia, in a third hysteria, and that there is no reason to separate hysteria arising from this cause from hysteria due to other causes, *e.g.*, moral emotions. The formation of a "traumatic neurosis" would be a false step in neurology, as it would be an artificial classification depending upon etiological factors only.

The facts are too numerous and too well authenticated to allow it to remain doubtful that the connection between traumatism and hysteria is one of cause and effect (in a predisposed subject) and not of mere coincidence. In further support of this may be adduced the fact that hysterical manifestations provoked by one traumatism, may be caused to disappear under the influence of another one. Cases of traumatic hysteria are of great importance from a medico-legal point of view. Railway accidents account for a good many of them, but other causes are equally efficient, *e.g.*, the fall of a mason from his scaffold, (21) of a coachman from his box, (21) a bronze-worker gives himself a blow on the fingers with his hammer, (21) a mother strikes her son on the ear. A burn, the application of a fracture apparatus, the wound inflicted in an operation, or even the force exerted in holding the patient in a particular position during the operation, have all been found effectual causes. Earthquakes, from the over-mastering terror they excite, are potent causes of hysteria; in the same way, lightening-stroke, or the fright produced by the near fall of a thunderbolt, may give rise to the neurosis. From numerous examples, M. Guinon concludes that the nature and degree of severity of the injury have little influence on the development of hysterical symptoms, and that any kind of injury whatever may provoke hysteria. For instance, in railway accidents—and the same remark applies to earthquakes—those who are most severely injured are not necessarily the most liable to consecutive hysteria. The contrary is almost true. The patient who afterwards becomes hysterical, is often able to reach his destination, either by another train, or sometimes even on foot. Meanwhile, the traumatism has indisputably a direct action on the determination of certain symptoms of local hysteria, *e.g.*, a fall on the shoulder has caused an hysterical paralysis of that arm. M. Pitres says, with regard to this point, that although there is a casual connection between a traumatism and the contracture which follows it, this contracture is not to be considered as a mere local effect of the traumatism, a so-called "local or peripheral hysteria." The injury has not caused the hysteria since that is present before and after the accident, it has

simply determined in the evolution of a malady already in existence a symptom which without the injury would never have appeared.

Prof. Pitres sums up the clinical characters of hysterotraumatic monoplegias in the following terms:—

(1) They occur indifferently in men and women, rather more frequently in the former.

(2) The patients are generally between the ages of 20 and 50 (adult life).

(3) They generally appear in persons predisposed to hysteria by hereditary influences, and who have previously suffered from neurotic affections. This, however, is not always the case, and a traumatic monoplegia may be the first manifestation of hysteria in a person previously free from it.

(4) The paralysis bears no relation to the severity of the injury. It follows severe or slight injuries, of local or general incidence, whether they are accompanied or not by nervous shock or emotional disturbance. A trifling accident may be followed by a paralysis which persists for years, whilst one that follows a serious injury may last for some days only.

(5) The paralysis may sometimes develop immediately after the accident, but usually comes on after an interval of hours or days.

(6) The paralysed muscles are flaccid; the electrical reactions are normal; the tendon-reflexes are normal, sometimes a little exaggerated. The muscular sense is lost. Rapid wasting often supervenes in the muscles affected.

(7) The skin of the paralysed limb is generally anæsthetic or analgesic. This anæsthesia may be limited to the limb, or extend to the corresponding half of the body, or may be general.

(8) The patients often, but not always, present the permanent stigmata of hysteria, *e.g.*, concentric contraction of visual fields—anæsthesia of pharynx. There are sometimes convulsive attacks and hysterogenic zones. They cannot usually be hypnotized.

(9) The duration of the paralysis is very variable. Cure is certain, but at an indefinite time. It often supervenes on a moral cause.

Toxic Hysteria.—In poisoning by lead, mercury, alcohol, bis sulphide of carbon, &c., hysterical manifestations are observed, more commonly in the chronic than in the acute forms. The recognition of the occurrence of hysteria under these conditions is of recent date. Although observations which proved that hysterical symptoms occur in plumbism, had been brought forward by

Petit (22) and by Maricourt, (23) who published a case of saturnine hemianæsthesia in which the absence of any organic lesion was confirmed by the autopsy, all authors—probably misled by the then prevailing idea that hysteria did not occur in the male—continued to attribute the anæsthesias of the general and special senses found in plumbism to organic changes in the nervous system produced by the poison, until a series of observations by many writers showed that these so-called saturnine anæsthesias could be made to undergo "transference" by the action of the magnet or faradic current. M. Charcot demonstrated clearly the hysterical nature of these anæsthesias, and, at the same time, the frequency of hysteria in men. (24) Poisoning by lead, alcohol, mercury, &c., may aggravate a pre-existing hysteria, or determine the advent of symptoms in a person predisposed to the neurosis, but hitherto free from symptoms. The poisons of the specific fevers, of diabetes, chlorosis, and syphilis may act in the same way. Plumbism, alcoholism, hydrargyrism, &c., may give rise to two groups of symptoms, the one directly due to the action of the poison; the other, of hysterical nature, and resembling those of ordinary hysteria. The former are directly produced by the poison, and soon cease when the patient ceases to be exposed to its action; the latter, once excited, run an independent course, and may disappear, (whilst the action of the poison still continues) being modified or suppressed by moral emotions or physical causes, just as in other forms of hysteria, or on the other hand, may continue after the poison has been eliminated from the system.

For instance, a painter who had had two years previously, an attack of lead poisoning was admitted suffering from colic, vertigo, headache, and paresis of the right upper extremity of 14 days' duration. There was a blue line on the gums; the abdomen was retracted and tender: two doses of castor oil removed the abdominal symptoms. The affection of the right arm, which consisted in general feebleness of movement, much more marked in the extensors than in the flexors (the wrist could not be fully extended), together with anæsthesia over the inferior third of the forearm, the dorsum and palm of the hand, and over the thumb, persisted. The diagnosis was doubtful but was cleared up by the application of a magnet to the limb when the motor and sensory symptoms disappeared completely in half-an-hour.

M. Pitres directs attention to the frequent occurrence of anæsthetic patches in poisoning by lead and alcohol. The clinical characters of this cutaneous anæsthesia are identical with that of

hysteria. This anæsthesia may be either complete or partial. Its distribution may be general or unilateral but most commonly occurs in disseminated patches. In the lower extremities it generally affects the feet and the legs enveloping them in anæsthetic boots, so to speak. In the upper extremities it often spares the palms and forms larger or smaller bracelets surrounding the limbs. On the trunk it frequently occurs "en cuirasse." The patches are often asymmetrical, their borders are clearly defined and their distribution shows no relation to that of the sensory nerves or the arteries of the skin. Neither the nutrition nor the temperature of the skin are affected and there are no vaso-motor disturbances. There exist also analgesia or anæsthesia of the mucous membranes of the mouth, pharynx, larynx, nose, ears, with general or partial impairment of taste, smell, and hearing and concentric contraction of the visual fields.

These anæsthesias correspond to those of hysteria in the absence of any organic lesions in the peripheral nerves or nervous centres, and in their tendency to disappear suddenly in consequence of moral emotions, electrisation, or application of metals (æsthesiogenic agents).

With regard to alcohol, Petit (25) who first makes mention of symptomatic hysteria, especially named alcohol as a cause of the neurosis. Nearly all authors consider that alcoholism is only occasionally a cause of hysteria, but M. Guinon observes that in making this statement most of them leave out of account those cases in which the neurosis formerly existed and was again excited into fresh activity by alcohol. With regard to one form of alcoholism Lancereaux states that the convulsive attacks from which absinthe-drinkers suffer are hysterical and not epileptic, and that he has never seen true epilepsy excited by drinking absinthe.

Two nervous disorders attributed to the action of mercury M. Guinon thinks are hysterical, namely, the sensory troubles and the convulsive and paralytic phenomena. The most important observations on hysteria provoked by mercurial poisoning are due to M. Letulle. (26)

M. Guinon prefers the terms hystero-saturnism, hystero-alcoholism, hystero-hydrargyrism for these affections, because no theory is implied by them and they do not therefore lead to misconception of the true nature of the cases. M. P. Marie (27) first pointed out the occurrence of hysterical symptoms in cases of poisoning by bisulphide of carbon.

In the second of his two works on the subject, Delpech (28)

divides the course of the symptoms caused by this form of poisoning into two stages. The *first* is characterized by cephalalgia, violence of character, epileptiform convulsions, sexual excitement and hyperæsthesias. The *second* by intellectual apathy, amounting even to hebetude, sad and terrifying dreams, cutaneous anæsthesias and affections of the special senses, impotence, and paralyses. M. Guinon shows, from the descriptions of recorded cases and from his own observation, that though neuritis occurs and causes certain nervous troubles, the nervous symptoms apart from these, are hysterical, and relies for proof of this on the characters of the anæsthesias, paralyses, affections of special senses, presence of hysterogenic zones and mental symptoms.

The first attack of hysteria may occur after or be provoked by anæsthetisation by chloroform, and this has occurred in cases where dread of the operation and the emotion produced by waiting for it can be eliminated.

The case is quoted (29) of a man who felt no disturbing emotions before the operation, which was a trivial one, in fact he wished for the operation and had already previously undergone without inconvenience a similar one, but on beginning to recover from the chloroform he had a violent hystero-epileptic attack lasting three quarters of an hour.

Besides symptoms due to organic disease of the central nervous system or peripheral nerves, poisons may evoke purely functional disturbances, in part belonging to hysteria. For Petit (30) hysteria is "symptomatic" of alcohol, lead, anæmia, &c. This theory of the "symptomatic hysterias," which is supported by Debove, (31) supposes that lead, alcohol, mercury, &c., are hysterogenic poisons, and may directly produce hysteria, complete in all its forms, even in persons not predisposed to it. They compare the "toxic hysterias" to the "toxic epilepsies," each of which has its characteristic clinical physiognomy. Fascinating as on many grounds this view is, it is not altogether satisfactory. A person in whom epileptiform attacks have been excited by lead-poisoning is not suffering from the same malady as another person who since infancy has been subject to epileptic fits periodically repeated without obvious cause. If the former is removed from the influence of the poison the attacks cease. Instead of speaking of a saturnine epilepsy we should say more correctly that he suffers from epileptiform convulsions of saturnine origin.

On the other hand—the "hysteria of toxic origin" theory—Charcot holds that poisons may aggravate existing hysteria, or excite its appearance in predisposed persons, but that they are incapable of creating it anew.

Apropos of two cases, one of lead, the other of alcoholic, poisoning, M. Charcot observes that the two patients were hysterical before being poisoned, and that some of the symptoms belong properly to hysteria while others depend on the super-added intoxications.

It is possible that hysteria may have been excited by the development of the intoxication, but this is not enough to sanction the names saturnine or alcoholic hysteria; we should rather say hysteria in a person poisoned by lead or alcohol.

M. Pitres, after the consideration of the foregoing theories, concludes that the distinction between true hysteria of toxic origin, and hysterical phenomena due to the action of poisons is difficult. An important difference is this, that true hysteria survives the exciting cause; but once aroused, whether by moral emotion, traumatism or poison, evolves itself independently of any new excitant; on the other hand, hysterical symptoms caused by intoxications remain subordinated and closely related in their development to the etiological circumstances which have aroused them. Traumatisms and poisons more frequently provoke hysteria in men than in women.

Hysteria is a pre-existing morbid susceptibility, in virtue of which various functional disturbances can be excited by etiological factors, which have in themselves only a secondary importance. Hysteria is innate, and the heredity creates the diathesis.

(The following observations on this question of intoxication, and on the traumatic neuroses, and on the mode of evolution of the symptoms are taken from M. Guinon's work).

Amongst the authors who do not admit "traumatic hysteria," some refuse to do so because, in their opinion, too great preponderance is given to the exciting cause. They do not allow that hysteria is a syndrome which becomes symptomatic of the action of such or such an exciting agent, and here they are perfectly right. But it is a long step from this to making neuroses afresh, and calling them traumatic, mercurial or lead neuroses. Although the same group of symptoms are produced in two individuals by a different cause in each, on this theory these symptoms would constitute a different disease in each of the two. This has been done for mercury and lead poisoning. In the case of the latter, A. Westphal (32) brings together almost all the nervous troubles which are observed as the result of chronic lead poisoning, under the head of "lead encephalopathy." Amongst his cases there are three well-marked examples of hysteria. Whilst quoting Charcot's opinions on the subject, he considers these cases to be ex-

amples of an "intoxication neurosis." M. Guinon thinks that this is a singular manner of regarding the facts, and it is better to multiply the causes of hysteria, than to give credit to such a vague term as "saturnine encephalopathy."

Others will not allow the name hysteria to be given to the nervous symptoms which follow traumatism or poisoning, because, although hysterical manifestations are present, yet other symptoms also exist which do not belong to hysteria. This is a clinical fact; and the key to the whole question lies in these complex cases, for by considering them closely, the objections brought against the theories of the French School may be refuted.

It has been said, but incorrectly, of the French School, that they hold that the nervous troubles that follow a traumatism are always due to hysteria, and to nothing else (33). But the French admit that traumatic agencies may provoke the outburst of many nervous affections, *e.g.*, paralysis agitans, epilepsy, senile tremor; they say, however, that because they have been excited by these agents there is no sufficient reason to put these cases in a category apart, and make of them a separate disease, a "traumatic neurosis." One might as well classify separately those cases in which white swelling of the knee arises from injury and those in which it appears to come on spontaneously. If ten cases of hysteria were taken, it would be impossible to say, from the actual state on examination—apart from the history—which were due to traumatism, which to lead, mercury, &c., the clinical entity is in each case identical.

M. Charcot has never held that hysteria is the only result of a traumatism; on the other hand, he has recognised the co-existence of hysteria and neurasthenia; and has pointed out (34) that this latter affection is equally frequent after traumatisms and railway accidents, and refers also to cases reported by Page (35). This coincidence of hysteria and neurasthenia is the key to the whole question; it is of common occurrence, but there is nothing to show that each of these affections remains autonomous. Just as in traumatisms, so in the case of other causes of hysteria, *e.g.*, intellectual overwork and lead poisoning, hysteria and neurasthenia may occur together.

One must bear in mind, that in a patient suffering from traumatic hysteria, other symptoms may be present, due to other causes. All the nervous symptoms complained of by an hysterical patient are not necessarily due to the neurosis. In a complex case symptoms, which may be due to an organic nervous affection, must be carefully weighed.

The exciting agents of hysteria are opportune causes of it, and nothing more; they cannot create hysteria anew, but only excite it in persons who are predisposed to it. This predisposition is hereditary, as Déjèrine points out (36). M. Guinon considers "that no convincing arguments have been advanced in favour of the existence of a 'symptomatic hysteria,' or of the division of the neurosis into several varieties of hysteria. The older and more rational hypothesis of a single and autonomous morbid entity—hysteria—is sufficient to explain all the cases, if it be granted that the exciting agents of the neurosis only play into relation to it the part of opportune (occasionelles) causes. This is easy to understand, if we consider that exciting causes behave, with regard to hysteria, just as they do in other maladies, in which their rôle of occasional cause is fully recognised and beyond dispute. It may be further remarked that these multiple or symptomatic hysterias, are all identical amongst themselves, and differ in no respect from the neurosis long described and considered as a fixed morbid entity." The exciting cause may impress a certain stamp on the hysterical manifestations. For instance, a woman, who had formerly suffered from compression paraplegia from caries of the vertebræ, when under the influence of the menopause, subsequently became hysterical, and developed an hysterical paraplegia closely resembling compression paraplegia. These phenomena are explained by unconscious cerebral influences acting in the form of auto-suggestion. At the same time no constant symptoms can be traced as occurring uniformly as the result of the action of one particular cause. All that can be said is, that speaking generally, "local hysteria," *i.e.*, monoplegias, monosymptomatic hysteria, occurs more often as the result of traumatisms than of other causes.

M. Charcot has pointed out the influence of falls or blows in producing monoplegia or contraction of the limb which has been the seat of the injury; for instance, a brachial monoplegia has followed a blow on the shoulder. Especially is this relation marked in cases of injury to the region of the eyeball, in which amaurosis, blepharospasm, contraction of the visual field have followed the traumatism.

The mode of development of hysteria under the influence of exciting agencies is twofold; it may either arise soon after the action of the cause or not till much later. The development may be best studied when the exciting cause has been in action for a brief and limited period. In cases of injury, for example, an uninterrupted chain of symptoms may follow the "nervous shock"

of the injury, or these symptoms may not appear until much later, and these cases are of interest as they are less known and less common than the first kind. M. Guinon gives the case of a man who, two-and-a-half years after a dangerous fall on a staircase, developed paralytic symptoms in the legs. The importance of these cases from a medico-legal point of view is obvious. Can we say that in a certain number of these patients, hysteria, provoked by the exciting cause, remains latent until paralysis, contractions, convulsions, draw attention to it? Some symptoms of hysteria, anæsthesia for example, may exist for a long time without the patient's knowledge. But generally, in cases of traumatic hysteria this does not occur; for the neurosis is then nearly always accompanied by a peculiar mental condition, which at once attracts attention. As a rule, hysteria is not latent in these cases.

Two or more causes may act together; for instance, in an alcoholic patient, hysterical manifestations—a case is given of paralysis of the forearm from a blow on the fingers in a drinker—may be excited by a slight injury, which would not have been sufficient to produce this result if the patient's powers of resistance had not been previously lowered by alcohol.

M. Guinon adduces many instances to show that several exciting agents of hysteria may unite, and produce an outbreak of the neurosis, when one acting alone, would not have been able to do so. There are cases in which hysteria existed at some former time, in which one cause to which the patient had been exposed was not sufficient to excite fresh manifestations of it, but in which another factor acting together with the first, was necessary to produce this result. He concludes that every individual presents a resistance peculiar to himself, to any particular cause of hysteria: one cause is more effective in his case than another. In each case there is always one cause which is more effective than any other.

All the exciting agents of hysteria, however varied and unlike each other they may seem to be at first sight, may be divided into two classes, according to the mechanism by which they arouse hysterical manifestations: (1) Those cases in which the symptoms of "local" hysteria predominate; (2) those in which the hysteria is general, without any striking local manifestations. In each group, all the cases which compose it are susceptible of the same interpretation. In "local" hysteria, the character of the paralyses and sensory affections indicates their cerebral origin; in it the immense majority of the symptoms are produced by auto-

suggestion. This auto-suggestion is derived most frequently from a traumatism, and after that from emotions. M. Charcot has shown the absolute identity there is between the phenomena of local hysteria, and those developed by suggestion in the hypnotic state. Now, paralysis during hypnosis may be brought about by verbal suggestion, for instance, by telling the patient he cannot move the limb, or by traumatic suggestion, by striking with a certain force the part that it is desired to paralyse.

Clinically, an auto-suggestion may occur in the same way. M. Charcot calls attention to what he terms "local shock." When a blow falls on a part of the body, it causes a sensation of numbness, of insensibility, or even a negative sensation of absence or loss of the part struck. In the healthy, this feeling soon passes off, but in a person predisposed to hysteria, it may intensify and undergo further development; and, as a consequence of this idea of absence of the limb, all idea of movement is totally abolished. In other cases of traumatic hysteria, the exciting agent, acting by a true traumatic suggestion, inhibits at once and persistently, motor representations. The mental phenomena that result from the action of the local shock dominate everything; the ideas of the existence of movement, and of feeling in the affected limb are instantly annihilated, and, if the patient's brain is suitably prepared for it, a psychical paralysis is constituted for a longer or shorter time. These cases are rare: as a rule, some time elapses between the reception of the injury, and the first appearance of the nervous troubles. The reason, why, in some instances, inhibition is instant, while in others, a certain lapse of time is required for the idea of paralysis to implant itself in the brain, M. Guinon thinks, lies in the difference of the cerebral state in different individuals, and not on the nature of the accident. He points out a striking parallel between these clinical conditions and the effects of suggestions made in different degrees of the hypnotic state.

When hysterical accidents follow immediately the action of the exciting cause, this is almost always a traumatism or an acute moral shock, such as terror. Terror may, indeed, be considered as a sort of sensation of powerlessness, either generalised, or localised in the lower extremities. In most cases of traumatism, terror and emotion must also be taken into account.

In the other group, the occurrence of an interval or period of incubation between the shock and the subsequent hysterical paralysis, explains the special form or the particular localisation that the exciting cause may give to the hysterical manifestations.

The local shock is the point of departure of the cerebral auto-suggestion ; the cerebral process, often an unconscious one, is, first, the loss of the feeling of the part, then the inability to move it. The arm feels heavy, flaccid paralysis, or stiff, paralysis with contracture. A phenomenon of some importance is the amnesia that often attends these accidents. In a large number of cases, with or without loss of consciousness, there is amnesia, not only for all the details of the accident, but for a certain period before it. The patients, having no notion of the details, and persuaded always of the great severity of the accident, imagine terrible consequences from it ; take their imaginings for reality, and, in perfect good faith, fabricate extraordinary accounts of the occurrence ; this amnesia has, therefore, often unjustly led them to be regarded as simulators. By a similar process of auto-suggestion, M. Guinon explains the action of the poisons of syphilis, lead, mercury, &c., in exciting hysteria. Auto-suggestion plays its part unconsciously to the patient. It may even be produced during sleep. Among the victims of accidents, terrifying dreams in which the accident is reproduced, are common. M. Charcot (37) and M. Féré (38) give instances of these dreams and their potency in auto-suggestion.

Prof. Grasset (39) recognizes the unity of hysteria, under a multiplicity of forms. He considers traumatic hysteria to be a distinct clinical type or species, with its own mode of evolution and special characters, many traits of which separate it from common hysteria. In criticizing the theory of auto-suggestion, he says that if we suppose that there is originally present a functional disturbance of the brain, capable by itself of bringing about a paralysis, to invoke the agency of auto-suggestion is to introduce unnecessary complication. In many cases of traumatic hysteria, the accident bears no relation to the succeeding paralytic or spasmodic phenomena. Will anyone seriously allege that in the cases where hemianæsthesia, affection of the special senses, or other "stigmata," follow a traumatism, they can have been produced by auto-suggestion? "Those manifestations of hysteria, which only develop a certain time after the traumatism, appear to be very difficult of explanation by suggestion. . . . To explain a post-traumatic paralysis separated from the traumatism by a long interval, it must be supposed that the injury provokes the auto-suggestion of the paralysis with this proviso, that the realization of the paralysis shall only take place after a certain time." He therefore limits the action of suggestion to cases where the hysterical symptoms arise from imitation (neuromimesis).

In other patients again the hysterical manifestations cannot be accounted for on this theory of auto-suggestion: amongst these, local hysteria is much less common, and the production of hysteria is due to a general alteration of nutrition which entails a failure of the whole organism, and especially of the nervous system, to perform their functions aright. Every exciting cause may bring about hysteria in this way, by interfering with nutrition. Especially is this the case in hysteria consecutive to acute or chronic general maladies, in diseases of the nervous system or genital organs, in physical or intellectual overwork, in the intoxications, in depressing emotions, and in prolonged grief.

Hysteria may appear during the exhaustion that follows upon an attack of one of the acute fevers; the nervous troubles consecutive to diseases of this class present themselves for the most part in the form of paralysis. M. Guinon says, with regard to acute rheumatism, that a sub-acute attack may appear to be more serious than it really is, because the pain and swelling of the joints determines hyperæsthesia of the skin and contractions of the muscles around them and there may thus arise a subsequent "pseudo-rheumatism" which may persist for months or even years and then disappear spontaneously (40). Diabetes may not only cause a recrudescence of hysteria but may evoke the symptoms for the first time. Syphilis may act in the same way. The various forms of anæsthesia which occur in cases of syphilis seem to be often due to consecutive hysteria (41). Malaria may impress a particular character upon hysterical manifestations, under its influence they take an intermittent form, recurring at regular intervals (42).

The old theories that placed the seat of hysteria in the genital organs are to-day universally discredited. Disease of these organs does not predispose more than that of any other organs to hysteria. Apart from this, from the importance attached to disturbance of the functions of the genital apparatus by women, a strong moral effect may be produced.

Severe hæmorrhages may be exciting causes, so may masturbation and venereal excesses from the nervous exhaustion they give rise to.

Hysteria may follow on physical or mental overwork, the latter is especially potent in adults, in children its influence is, to say the least, doubtful. Children cannot be forced to perform an injurious amount of intellectual labour. If children are given an excessive quantity of work, they do what exceeds their intellectual powers like machines, materially, so to speak; not seeing the

immediate interest or object of the task allotted to them, they are incapable of voluntarily exciting that state of over-action or excessive tension of the cerebral tissues necessary to produce an amount of work beyond their powers. If they suffer it is from too long detention in class-rooms and from want of sufficient exercise and fresh air.

Hysteria may also be excited by nervous diseases, tabes dorsalis, Friedreich's disease (43), idiopathic muscular atrophy, and pressure paraplegia from caries of the vertebræ.

In men hysteria affects especially the lower classes, whether they are unskilled labourers, or engaged in some special occupation; very often affected are stokers, engine-drivers, divers, tobacco employés, sailors, and soldiers. In the last the severe type of hysterical convulsions, absolutely analogous to that which occurs in women, has been described (44).

M. Colin observed that cases of hysteria are not at all common amongst the strictly criminal classes, and from the point of view of number, there is in them no difference between the sexes. Hysteria is much more frequent amongst tramps, beggars, and vagabonds (45).

M. P. Marie has published statistics to show that compared with men, the subjects of hysteria in women stand higher in the social scale. M. Gilles de la Tourette thinks that such statements are very difficult of exact proof. If hysteria is more common in town than in country women, it is to be attributed to the fact that the exciting causes, particularly the forms of intellectual and emotional excitement, which make so much more profound impression on the female than on the male nervous system, are more common in town life. The opinion of Briquet (46) is quoted with approval that "whatever tends to weaken the general health to a certain degree increases at the same time the excitability of the nervous system. Hence it follows that the sedentary professions, those which entail long hours in workrooms, and those which afford a precarious livelihood, in so far predispose to hysteria, whilst on the other hand the professions which necessitate movement and open air exercise, and afford at the same time sufficient means of sustenance will in equal degree be hostile to it.

Continence or excessive sexual intercourse seem *per se* to have little effect in evoking hysteria, but the hard conditions of a prostitute's life place her under favourable circumstances in other respects for the development of the neurosis.

All races are subject to hysteria. Of the white races, the Jews are especially prone to suffer from it. M. Gilles de la

Tourette says that the many observations that have now come from authors of both countries and from the large international practice of M. Charcot permit us to say that hysteria like other nervous diseases is exactly the same in Germany and in England as in France, in contradiction of the statements that have been made that hysteria is more rare in England and Germany and occurs in a less severe form than in France, and that the descriptions of M. Charcot only apply to the latter country. He quotes some observations of M. Aruch at the Veterinary School of Milan in which various nervous symptoms in three dogs, namely, paresis, paraplegia without affection of bladder or rectum, anæsthesia, aphonia, and convulsive attacks without loss of consciousness were attributed to hysteria, no lesions being found at the autopsy which could account for these symptoms.

The only way for us to get a thorough understanding of hysteria, is to study it by the experimental method. The classification of cases into acute and chronic is unsatisfactory on account of the variable and fortuitous course of the malady, regulated as it is by external agencies. The division into acute and chronic moreover ignores the latent predisposition always present. Based on the nature of the predominant symptoms in each particular case, hysteria has been classified as (1) convulsive or non-convulsive, (2) peripheral or visceral, (3) mono- or poly-symptomatic. These divisions may be adopted for convenience if it is always remembered that they do not correspond to different forms of the disease. Any case by virtue of the hysterical diathesis may at one time or another present any of the various symptoms. It is exceedingly rare for one symptom to exist alone. Alongside of the symptom that has attracted the patients' attention, the physician connotes the presence of latent symptoms or stigmata, whose concomitance is indispensable to the diagnosis. Classifications based on age and sex have no value. To sum up, hysteria is one and indivisible. Under, the manifestations which vary in accordance with the infinite variety of contingent circumstances, lies always the same disease as the true source of them.

In hysteria, as in all general diseases, there is a ground work on which the symptoms which constitute the affection evolve. This ground work "the general hysterical state" is formed by the union of a great many elements. It is not always complete, many links in the pathological chain may be absent at the time of observation, but one character, *tenacity*, is common to all, and they have therefore been named by M. Charcot the "permanent

stigmata" (47). Their study is of the first importance in diagnosis, for they enable one to affirm the existence of hysteria when the more striking phenomena, which are often temporary, are absent, and a thorough acquaintance with them is indispensable for a sound interpretation of these latter symptoms.

The history of the discovery of anasthetic patches in hysterical subjects is very interesting. M. Pitres recalls that when the magistrates who were investigating a case of witchcraft examined the accused, they began by trying to establish moral proof of demoniacal possession by questioning him on his family history and his antecedents, and endeavoured to get him to confess his commerce with Satan, and then proceeded by means of pricking him with pins (the accused being blindfolded) or sharp splinters to determine the presence of anasthetic areas in the skin, which were called stigmata diaboli or witch-marks. To the discovery of these stigmata great importance was attached as a proof of the transport of the accused to the witches' "sabbath," and they were attributed to direct contact with the devil. Readers of contemporaneous accounts of trials for witchcraft will remember the importance attached to the presence of wens or moles and of small areas into which a sharp needle or awl could be thrust even to the bones without pain and at the same time without causing bloodflow. The doctors of the time shared in these superstitions, and Jacques Fontaine, "conseiller et médecine ordinaire du roy et premier professeur en son Université de Bourbon en la ville de Aix," wrote a book on "Witch Marks and the real possession by the devil of the bodies of men." (48) He thought that these "dead-anæsthetic patches" were made with a hot iron and a certain ointment, and accounted for the absence of any scar by supposing that "Satan was such an accomplished operator that he could apply the iron to the body without producing a scar." To day the physician proceeds in the same way as the judges of the middle ages; when he suspects a person of hysteria he begins by trying to obtain moral proof of the existence of the neurosis in the morbid tendencies—hereditary and personal—of the patient, and investigates the nature and character of past and present symptoms. Lastly, he searches for impairment of the general sensibility or of the special senses, which are now considered as "marks of hysterical possession" or "hysterical stigmata."

It is a remarkable fact that when witchcraft ceased to be believed in the existence of these anæsthetic patches was completely forgotten, and it is hardly fifty years since anæsthesia was recognised by doctors as common in hysteria. The first regular

observations were those of Piorry in 1843. Briquet in his work on hysteria in 1859 investigated and described them with remarkable thoroughness and perspicacity, but hysterical anæsthesia was not generally recognized until the popularization of the subject by M. Charcot in his observations on hysterical hemianæthesia.

Varieties of cutaneous anæsthesia in hysteria.

(1) Total affecting all forms of sensation.	(a.) Complete—anæsthesia proper. (b.) Incomplete—hypæsthesia.
(2) Partial, affecting certain forms of sensation only.	(a.) Loss of sensation to pain only—analgesia. (b.) Thermo-anæsthesia, loss of sensation to temperature but not to touch or pain. (c.) Loss to touch and pain with preservation of temperature sense. (d.) Loss of sensation to electrical stimulation only. (e.) Preservation of sensation to electrical stimulation alone with loss of other forms of sensation.

In testing for sensory defects, an assistant, with his finger and thumb, closes the eyelids of the patient, this procedure avoids all source of error from the slipping of a bandage. For tactile sensibility it is sufficient to touch the skin with the index-finger, by using greater or less force, the complete or incomplete loss of tactile sensation is judged. The patient counts aloud 1, 2, 3, 4, &c., as soon as he perceives the contact, and thus an idea can be formed if perception is normal or retarded. Objects of different form or weight are placed in his hands, in order to test his perception of form and weight. M. Charcot always employs only the simplest procedures, and never uses complicated æsthesiometers. It is necessary to carry out the investigation quickly as otherwise one may sometimes provoke the phenomena of transference. To test for analgesia a pin, with a fine sharp point is used; it is necessary to make the pricks sufficiently profound, and to go over an extensive surface of the skin, or at any rate to prick the skin in several places widely separated from each other. The results may be then, for convenience, marked out on a chart.

Pitres has shown that the nerve-trunks are insensible. He calls attention to the theoretical interest of these observations. "They prove, unless I am deceived, that hysterical anæsthesia does not result from a functional inertia of the terminal extremities of the sensory nerves. For if the seat of this anæsthesia were peripheral, the direct irritation of the nerve-trunks should arouse sen-

sations of pain in the nerve-centres which remain intact. Since it is possible to strike and prick the nerves of the limbs without provoking any sensory reaction, the seat of the anæsthesia must be above the nerves, and as there are only the nerve-centres above them, the determining cause of hysterical anæsthesias must be sought in these centres."

In the healthy, a violent blow on the epigastrium is sometimes immediately fatal, a lighter one gives rise to a passing syncope, this effect M. Pitres has never observed to be produced in an hysteric. He gives an example where forcible blows on the epigastrium of a patient caused her to feel a sensation of well-being, and she smiled at blows which would have made a strong man faint. He considers that the abdominal and thoracic viscera are anæsthetic in hysteria. The bones, ligaments, and articulations are also frequently insensitive.

For the investigation of thermo-anæsthesia, M. Charcot uses a thermometer with a flat bulb, the lower part of the tube and the bulb being enclosed in two metallic cylinders. The outer cylinder glides over the other, and can be raised in order to see that the apparatus is in good order. The inner cylinder is filled with copper filings. The outer cylinder is warmed in the flame of a spirit lamp, care being taken that the heat is not too suddenly applied, and that the index of the thermometer does not pass beyond 100° C. The position of the index being observed, and the base of the outer cylinder applied to the skin, accurate observations on the temperature-sense can be made.

As a general rule, anæsthesia involves all forms of sensation in the affected areas, but the different forms are frequently dissociated, one of the most important varieties being the simulation of the affection of sensation met with in syringomyelia. M. Charcot found that of seventeen cases, in eleven the anæsthesia was of the common type, in two, sensibility to touch and pain was preserved, but the temperature-sense was lost, and in four, the condition corresponded exactly to that described in cases of syringomyelia; *i.e.*, tactile sensation was normal, whilst sensibility to pain, heat, and cold, was either absolutely lost or very greatly deficient. Isolated loss of the tactile sense does not seem to occur.

Distribution of cutaneous anæsthesia.—General anæsthesia of the skin is rare as a lasting symptom, (one per cent. of cases): as a temporary symptom it is more common, often appearing for some hours after a convulsive seizure; for instance, it occurred after a convulsion in a patient generally hemi-anæsthetic, and

then gave place to hemi-anæsthesia again. In such cases there is abolition of the special senses, complete amaurosis, deafness, and absolute loss of taste and smell.

In the most common form, the distribution of the anæsthesia is unilateral. In some cases the hemi-anæsthesia is rigorously unilateral, in others, the sensory disturbance is not uniform over the affected side.

Cutaneous hemi-anæsthesia is accompanied with sensory disturbances of the mucous membranes, special senses and deep tissues, *predominating* on the side of the cutaneous affection; predominating but not rigorously limited to this side. M. Gilles de la Tourette has never seen a case, and he quotes some recent observations of Thomsen and Oppenheim to the same effect, in which the skin, mucous membranes, and special sense organs, were simultaneously affected on one side of the body, whilst on the opposite side they remained absolutely normal. When the field of vision is contracted on the anæsthetic it is always a little contracted on the other side also. Hearing is often equally affected or equally spared on both sides. Speaking generally, the parts of the mucous membranes which border on the skin, are similarly affected to it. On the other hand, the mucous membrane of the larynx, of the epiglottis, of the posterior part of the nasal fossæ, of the tympanum and the integument of the bony portion of the auditory canal, preserve some independence relatively to the cutaneous anæsthesia, and are completely anæsthetic, hypoæsthetic, or normally sensitive over their whole extent. This point is of practical importance, for according to Lori and Ott, anæsthesia of the deeper mucous membranes, especially of the larynx and epiglottis, is always exactly unilateral in cases of hemianæsthesia due to an organic intra-cerebral lesion.

(*c*) The anæsthesia may be distributed in disseminated islets, of which it is impossible, from their variability, to give a methodical description. The anæsthetic patches occur indifferently on the head, trunk, or limbs. Their number and size are very variable. Their form does not correspond to the anatomical distribution of the sensory nerves, nor to that of the blood-vessels of the skin. More or less extensive anæsthesia of the mucous membranes, and of the special senses is concomitant, and there is generally some loss of muscular power on the side on which the loss of sensation predominates.

Hysterical anæsthesia is often found in clearly circumscribed areas, the knowledge of which we owe to M. Charcot, who showed that, in male hysteria, in certain cases of brachial mono-

plegia anæsthesia was superimposed on the paralysis, and in such a way, that at that time, when syringomyelia was still imperfectly known, its distribution alone was sufficient for diagnosis. In a case of paralysis of one upper extremity, the shape of the anæsthetic area recalled one of those pieces of sixteenth-century armour designed to protect the shoulder—and also affected almost the whole arm. In paralysis of the shoulder only, the anæsthesia is limited below by a horizontal plane perpendicular to the axis of the limb, situated at two fingers' breadths above the elbow in front, and posteriorly passing above the olecranon. In paralysis limited to the elbow, the lower line of the anæsthesia is circular and horizontal, situated about two fingers' breadths above the wrist, in a plane perpendicular to the longitudinal axis of the limb. M. Charcot concludes that "this arrangement in *geometrical segments* delimited by circular lines in planes perpendicular to the long axis of the limb, represent, at least for the limbs, the type of anæsthesias of cortical origin, whatever the lesion may be. Only in hysteria this characteristic will generally be much more marked and easy to recognize than in organic focal lesions . . ." (49) It is unnecessary to add that the lower extremities may be affected in the same way as the upper. Hysterical anæsthesia does not follow the distribution of the cutaneous sensory nerves.

Anæsthesia may appear suddenly, may undergo many sudden modifications, or may spontaneously disappear: its extent and severity in any particular case are often in correspondence with the severity and extent of the other symptoms. It is valuable in diagnosis, because of the impossibility of its being successfully simulated.

The principal symptoms associated with hysterical cutaneous anæsthesia are: (i.) abolition of the superficial reflexes. M. Pitres has seen the abdominal reflex preserved, though feeble, on the anæsthetic side in cases of hemi-anæsthesia, he has also seen it absent on both sides in hysteria with diminished sensibility. Both he and M. Gilles de la Tourette agree "that there is no reason to attribute to this symptom the diagnostic value which Rosenbach's observations seemed to give it." This author stated (50) that the abdominal reflex is lost in certain cases of cerebral lesions, but is always preserved without alteration in hysteria.

(ii.) Preservation of the organic reflexes, *e.g.*, reddening of the skin by a mustard leaf, or the raising of a blister by a vesicatory. The pupil also dilates on sensory stimulation to a sharp pinch or

prick, even of the anæsthetic part of the skin. Rosenberg's observation, that in cutaneous anæsthesia the insensitive parts readily take the temperature of the surrounding medium, does not hold good for hysteria.

(iii.) Very remarkable as contrasted with anæsthesia of organic origin, is the absence of disagreeable subjective sensations, or of notable disability to perform the common actions of every-day life.

M. Pitres says that "he has closely questioned the hysterical patients under his observation and he can affirm that they were for the most part unconscious of their sensory troubles (anæsthesia). When pressed some of them said that they had remarked that they did not feel any pain when accidentally pricked or injured in particular parts. Others alleged that the anæsthetic limbs were less adroit at work, which demanded precision of movement. One hemianæsthetic patient said that she perceived the temperature of the water of a bath on one side of the body only. None of them suffered from the presence of uncomfortable or painful sensations, (numbness, tingling, prickings, pins and needles, &c.) in the anæsthetic areas of the skin. This is an important peculiarity, for generally anæsthesias dependent on organic lesions of the spinal cord or peripheral nerves are accompanied by subjective sensations and functional troubles which strongly attract the attention both of the patient and of the doctor. Hysterical anæsthesia, on the contrary, is a latent symptom that the physician must seek out by a regular and methodical exploration."

M. Gilles de la Tourette agrees with Prof. Pitres as to this, "although he has sometimes noted subjective sensations of numbness, and formication, and sometimes more or less acute pain in the anæsthetic limbs. A patient with total general anæsthesia avoided, without knowing the reason, walking in the dark. The difficulty she found was explained by the fact that when she closed her eyes she could no longer stand because she did not feel the contact of the ground. A strong and vigorous man walked about the courts of the Salpêtrière during very cold weather in his shirt sleeves without appearing to feel cold. This singularity gave him no uneasiness and he was altogether unaware of his thermo-anæsthesia."

Hysterical patients with hemianæsthesia complain most frequently of feeling the whole side of the body heavy; the arm of this side is more awkward and weaker than ordinarily. "With some reservations we do not hesitate to conclude, that in the

great majority of cases anæsthesia requires to be sought for and is almost always first recognized at the medical investigation.'

Professor Pitres does not agree with Briquet's statements (*op. cit.* p. 283) that "the temperature of the anæsthetic skin is lowered one or two degrees Centigrade," and the circulation through the capillaries is slowed. On the contrary, he finds that neither the circulation nor the temperature is altered on the anæsthetic side. Sweating is as easily induced by pilocarpine, the skin is reddened by irritants, becomes inflamed and suppurates exactly in the same way in the anæsthetic as in the sensible parts. He concludes that there is no reason to believe that hysterical anæsthesia depends on a local disturbance of the circulation.

It appears to be certain however that there is a real ischemia in the anæsthetic regions, shown by the fact that the anæsthetic skin rarely bleeds in the site of pricks. M. Charcot relates some instances of this kind (51). "Some leeches having been applied to a patient with hysterical hemianæsthesia, I remarked that the bites bled with great difficulty on the anæsthetic side, whilst on the healthy side, there was the usual amount of bleeding. Grisolle, a very skilled and exact observer, noticed the same thing. This ischæmia, which by the way very rarely attains to this degree, explains certain reputed miraculous circumstances. For example, in the epidemic of St. Médard, it is said that sword-cuts did not give rise to hæmorrhage in those affected with convulsions."

M. Pitres thinks that the contact of a foreign body excites a strong contraction of the cutaneous vessels before the blood has time to flow. When the anæsthetic skin is pricked, a pale areola, soon surrounded by a wider zone of redness, appears around the pin-prick, and quickly gives place to a raised papule or minute wheal, which persists for half an hour to an hour or longer. After the papule has formed, a little serous fluid may flow from the orifice of the prick. There is thus produced an immediate contraction of the vessels in the site of the puncture, and a localized œdema limited to this spot.

He goes on to say that this "vascular hyper-excitability" which prevents the flow of blood after a prick, is sometimes general, and not limited to the anæsthetic region.

If, however, a blister, which always reddens the skin over the insensible areas, be first applied, pricks over the reddened surface bleed freely, though sensation often remains unaffected.

In commenting on these observations, M. Gilles de la Tourette states that in a series of experiments which he made with M. Cathelineau on ten hysterical patients, for the purpose of

studying the composition of the blood in hysteria, they never noticed that the wet-cuppings bled differently on the anæsthetic and on the sensitive side of the body. The simple preparatory application of the cupping glass did not play in this case the same rôle as the blister described above, for it reddened equally the skin of each side. (52)

M. Guichon has further shown (53) in opposition to some observations of M. Teissier of Lyons, that subcutaneous absorption takes place as well on the anæsthetic as on the sound side.

M. Pitres has found in some cases a form of paræsthesia in which the contact with the skin of substances, which commonly only cause a sense of contact, gives rise to intensely painful sensations: he gives the name of haphalgesia to this symptom. He also relates two cases in one of which if a piece of copper, brass, gold, or silver were placed in the hand on the anæsthetic side of the body, a strong contraction of the limbs resulted, the patient became agitated and after a few seconds was convulsed. In the other contact with silver caused a burning sensation, which rapidly increased until it became so acute as to cause loud cries or even a convulsive attack. Other substances were inert. The experiments made were so carefully guarded that intentional deception would seem to have been impossible. M. Gilles de la Tourette has seen one such case only: this patient could not bear the contact of gold, and could not wear ornaments of this metal on account of the burning sensation produced by them.

Anæsthesia, though very frequent, may be absent in cases of confirmed hysteria. M. Charcot teaches that in children especially hysteria presents itself in a monosymptomatic form, paralysis, contracture, rhythmical chorea, or other symptom without any affection of general or special sensation. Of 40 adult patients M. Pitres found two free from any sensory defect.

Out of 235 patients Briquet observed general anæsthesia in 4, hemi-anæsthesia in 93, and anæsthesia in patches in 138. Hemi-anæsthesia occurs more often on the left than on the right side.

In the muscles there is loss of pain on pressure. The muscle, which is insensitive to pressure between the fingers, is readily excitable on percussion or electrical stimulation, in other words, in spite of its analgesia, its contractile power remains intact. There is loss of the muscular sense and therefore some uncertainty of voluntary movement with the eyes closed, and of the ability to distinguish between different weights; there is also loss of the sense of fatigue which normally follows on sustained muscular effort.

The loss of the sense of muscular contraction may be a cause of awkwardness or uncertainty of movement but not of paralysis. In other cases again the patients can perform any movement with their eyes open, but when the eyes are closed they are unable to accomplish any action. Lasègue (54) showed in 1864 that if the sense of touch were utilized, *e.g.*, by placing the anæsthetic hand on the unaffected skin of the head, movements of the hand could be carried out with the eyes closed, so that touch could thus be substituted for sight. Professor Pitres in a case where the left arm was anæsthetic, found that the grasp of the left hand was nil with the eyes closed and 50 kilos with the eyes open. If his hand was placed—the eyes being shut—on the table and he was told to move his fingers as if playing the piano, he could not do so, but if the hand was placed on the thigh he moved the fingers at once. He shows also that regular or rhythmical movements if begun with the eyes open can be continued after they are closed, but if stopped cannot be initiated anew without opening the eyes, and that bilaterally associated movements can be started and carried out with closed eyelids, *e.g.*, the patient mentioned above could perfectly perform the movements of the arms for swimming when he was entirely unable to move the left arm alone without the aid of sight. He considers that the phenomena described by Duchenne under the name of paralysis of the muscular sense depend chiefly on a partial disturbance of motor innervation. This disturbance, of central origin, can be corrected by certain sensory impressions and by several modes of bringing into action the motor centres. It is not simply an affection of the muscular sensibility. Anæsthesia of the muscles may be one of its constituent elements but its real origin is outside the muscles, and even outside the centres of sensory perception.

The sense of position of the limbs may be preserved in the absence of muscular sensation, of the sense of fatigue, and of that of contraction of the muscles; these senses do not therefore depend the one on the other. As a general rule however, the sense of position is perverted or abolished when there is profound anæsthesia of the muscles and skin. Not only the sense of position of the limbs but that of the size and weight of objects handled may be lost. If there is general anæsthesia the patient may be lifted out of his bed on to the ground, if his eyes be closed, without his perceiving the change in his position. If he is suddenly ordered, whilst walking, to close his eyes, he will fall heavily to the ground.

Normally we are conscious of the existence of the several parts of our body, this consciousness being the sum total of the numer-

ous sensory impressions of all kinds which reach the brain from all parts. It is a curious fact that hysterical patients, in spite of complete anæsthesia of the skin and deeper tissues, often retain the notion of the existence of their members. Only in certain cases of both motor and sensory paralysis is the sense of the existence of the limbs absent. M. Pitres thinks that further observations are required to determine whether the cause of this loss lies in the nerves or in the sensory centres.

Hysterical anæsthesia of the mucous membranes and of the special senses.—The most common forms are total anæsthesia, analgesia or hypoæsthesia; haphalgesia may also be observed. The patients are generally as unconscious of anæsthesia of the mucous membranes as of that of the skin when they first come under observation. The "organic reflexes" are preserved; an irritant placed in contact with the tongue, conjunctiva, or nasal mucous membrane provokes a flow of saliva, tears, or nasal mucus respectively. These affections of the mucous membranes may be the first or the only hysterical stigmata to appear. When cutaneous anæsthesia is general, the mucous membranes and special senses are affected on both sides: when it is unilateral, on that side only.

The loss of the special sense is added to that of the general sensibility of the part; that is to say, in the majority of cases the anæsthesia affects not only the special sense organ itself, but also the mucous membrane or skin which covers it.

This rule especially holds in hemianæsthesia. There, as M. Charcot has shown, the special senses are abolished or obscured on the anæsthetic side, and as a rule on that side only. The eye is an exception, for generally the concentric contraction of the visual field is bilateral. At the same time it is always greater on the anæsthetic side. In these cases also the insensibility of the buccal mucous membrane may be complete on both sides.

Anæsthesia of the anus and of the urinary passages is, according to Briquet, very rare, much more so than that of the mucous membrane of the genital organs. In the vagina the anæsthesia is often limited to one side of it.

Unilateral loss of taste is common, and may exist as an isolated "stigma." M. Lichtwitz (55) has found in some cases that together with partial loss of taste on parts normally gustatory, taste was present on other parts of the buccal mucous membrane, *e.g.*, the palatine vault, which have, as a rule, no such function.

He used the galvanic current as the test in his researches and

states: (i. that in six cases of hysteria the "field of taste" (gustatory area) was diminished or abolished; (ii.) the gustatory area determined in this way was either of the same extent or smaller than that for sapid substances; (iii.) there seemed to be no relation between taste and general sensation as determined by galvanism.

M. Pitres divides the patients from this point of view into four classes:—

(i.) Those in whom taste and common sensation are lost together over certain areas of the tongue.

(ii.) In others taste is lost whilst common sensation remains intact.

(iii.) Sometimes taste is preserved over parts that are insensible to pain and temperature.

(iv.) In some there are areas of tactile anæsthesia over the tongue, and of loss of taste, the limits of which do not correspond.

M. Charcot and M. Gilles de la Tourette have found, however, that in the great majority of a very large number of cases examined by them, loss of common sensation and of the special sense are superimposed upon one another, not only in the case of taste but of the other special senses.

The comparative frequency of affection of taste in hysteria, as compared with other nervous diseases, renders it a valuable symptom in diagnosis.

The frequency of anæsthesia of the larynx is very variable in hysteria; according to Thaon (56) one-sixth of hysterical patients are affected with it. It affects both sides of the larynx, and is never unilateral: it is often associated with paralysis of the vocal cords.

As regards the nasal mucous membrane, M. Lichtwitz (57) says, that it is, perhaps, of all the mucous membranes, the least affected with anæsthesia. His conclusions are opposed to those of Briquet, which have met with general acceptance. Briquet wrote: "The circumference of the anterior orifice of the nasal fossæ, and all parts of these cavities which are accessible to instruments, may be pricked with a pin, without the patient feeling it." At the same time, M. Lichtwitz has very frequently observed the presence of spasmogenic or hyperæsthetic areas in the nasal fossæ. It is difficult to see why the nasal mucous membrane should be exempted from the general rule that M. Charcot teaches, namely, that the mucous membranes participate in the anæsthesia of the affected side in hysterical hemianæsthesia, the affection of the special senses being superimposed on that of common sensation.

In eleven cases, Lichtwitz found unilateral anosmia in four, total anosmia in one, bilateral loss of smell for certain odours only in one, and once a pronounced diminution of smell in the left nostril only. Several patients found asafœtida not disagreeable —one was hyper-sensitive to all odours.

Hysterical deafness may be absolute; coming on suddenly it may last for days or weeks, and as suddenly disappear, but in most cases it is partial only. Generally this deafness is associated with loss of common sensation over the external auditory meatus and tympanic membrane, but this is not always the case.

The reader is referred to Walton's observations on deafness in hysterical hemianæsthesia in BRAIN, vol. i., pp. 458-472, 1883.

The deaf ear is often affected with tinnitus. Sometimes the sounds in the ear are so loud and persistent as to resemble those of Ménière's disease; and then, should an attack with vertiginous symptoms come on, the difficulty of diagnosis is great. In these cases, the auditory meatus or the auricle, is probably the seat of an hysterogenic zone.

It is not doubtful, M. Gilles de la Tourette remarks, that the shocks sustained by the organism in general, whether the patient be previously exempt from the neurosis or not, have a special influence on the genesis of anæsthesias of the skin or special senses, whether these are extensive, total, in patches, or as is generally the case, unilateral. Local injuries have a direct action in the production of geometrically-distributed cutaneous anæsthesias. M. Charcot relates the case of a woman, who after giving a box on the ear to her child, was taken directly after with hysterical paralysis of the hand, with the addition of anæsthesia. In another case, the anæsthesia, which affected the paraplegic lower limbs, was exactly limited by a circular line at the level of the imaginary passage over the body of the wheels of a carriage which had knocked the patient down. (58). In such cases, the topographical distribution of the anæsthesia seems to be determined by the paralysis. Very often the anæsthesia persists after the cure of the paralysis, an important element in the prognosis, for so long as the anæsthesia remains, the paralysis may return.

As regards the mucous membranes, and especially those which cover the special sense organs, in the majority of cases, anæsthesia supervenes without any recognizable direct cause, but in a fair number of cases, a relation between the exciting cause and the anæsthesia can be traced. For instance, a blow from the fist, or with a whip, may directly give rise to amblyopia. Note, however,

that local causes (traumatisms) seem to play no part in the genesis of gustatory troubles. The influence of all causes is limited to determining the localization of sensory symptoms on the common ground-work of the neurosis.

The data as to the mode of onset of hysterical anæsthesia are incomplete, but it may probably be either sudden or gradual. Anæsthesia may replace hyperæsthesia or *vice versâ*. We have fuller knowledge as to the course of hemianæsthesia.

It is one of the most fixed (permanent) of hysterical stigmata, and this applies especially to hemianæsthesia with the corresponding defect of the special senses. In an hysterical woman, æt. 65, at the Salpêtrière, hemianæsthesia still persists after thirty years, though all other signs of the neurosis have gone; and in another, in whom the diagnosis was confirmed at the autopsy, it existed for more than forty years.

With regard to the occurrence of anæsthesia, cutaneous and of the special senses, at different ages, M. Charcot teaches that it belongs especially to the adult age, both in men and women. In the old, the symptoms of hysteria gradually die away, but anæsthesia may persist as the two cases just quoted show. On the other hand, in children sensory troubles are more rare. They may be present, but never to any great extent. Some disturbance of taste, of hearing, a slight retraction of the visual field, are the forms most often met with in them. In accordance with this the hysteria of children is less severe and is capable in a large number of cases of absolute cure, whilst on the other hand it is especially obstinate and refractory to treatment in the adult. Hence the importance with regard to prognosis of the sensory stigmata; their presence indicates a severe type of the disease.

Briquet and Duchenne found that anæsthesia could be caused to disappear by faradisation.

The researches of MM. Charcot and Burq showed that by the contact of certain metals, or of a magnet, by the use of electrical currents, or of mechanical vibrations, the sensibility of the anæsthetic parts could be restored. They found an idiosyncrasy to the action of certain metals in particular patients; that in one anæsthesia disappeared under the influence of iron or copper, and in another under that of gold or zinc. At the same time the phenomenon of "transference" was discovered, that is to say the anæsthesia under the action of the "æsthesiogenic agent" left the side of the body first affected and passed to the other one. Subsequently many other substances were found to act in the same

way. M. Pitres shows by the aid of experiments with mercury that it is not necessary for the metals to be in a solid state. Mercurial vapour and liquid mercury were equally efficacious. Of the four gases, hydrogen, coal gas, oxygen, and carbonic acid, the former only was effectual.

The following short account of the phenomena of metalloscopy is by M. P. Richer (59). "Suppose a patient to be affected with hemianæsthesia. Several metals are applied to the cutaneous surface without affecting the anæsthesia; at last that one is applied—gold for instance—to which the patient is sensitive. Two or three twenty-franc pieces—one is sufficient—are placed on some part of the anæsthetic side of the body, say on the forearm. In a few minutes the patient feels a sensation of heaviness and tingling in the limb. If then, the eyes being closed, he is pricked with a pin in the immediate neighbourhood of the coin, he feels pain; if he is pricked at a distance of some centimetres from it the pricks are no more felt than before. A little later this area of sensibility extends, and in extreme cases it may happen that the anæsthetic side wholly recovers its sensation momentarily. We say momentarily, because if the experiment is prolonged the anæsthesia re-appears, beginning in those parts in which it disappeared last, and the patient returns to his former state. The total duration including the disappearance and re-establishment of the anæsthesia, varies with the surface-area affected, lasting, from half a minute to three-quarters of an hour or more."

Accompanying the return of sensation is an increased supply of blood: pricks cause bleeding now whereas they were before bloodless, there is a rise of temperature in the part, and increased muscular power.

Vice versâ by applying the metal to the sound side of a hemianæsthetic patient, the anæsthesia may be caused to transfer itself to this side. If the experiment is prolonged however, the side first affected again becomes anæsthetic.

Consecutive oscillations. Three varieties of consecutive oscillations are described by P. Richer:—

(i.) The patient wholly recovers sensibility for a time.

(ii.) He remains in a state of "transference," *i.e.*, if he had left hemianæsthesia before the experiment, he has, for some instants, right hemianæsthesia.

(iii.) Finally he relapses into the state prior to the experiment.

M. Pitres thinks that it is probable that "transference" is simply an exaggeration of a well known physiological phenomenon. In 1854 Hoppe (60) observed that if the thumb and index finger

of one hand are sharply pricked there is a marked diminution of sensibility on the symmetrical points of the other hand. Other authors have made similar observations, notably, those cases in which transference of the motor areas of Jacksonian epilepsy by the application of a circular blister above the point of origin of the primitive area has occurred. These observations show that transference is not such a strange phenomenon as it appears at first sight, but is found in an attenuated form under normal and pathological conditions altogether independent of hysteria.

It is certain that the æsthesiogenic agents, metallic or non-metallic, do not produce constant and uniform effects on all subjects. Every patient is influenced by such or such an agent to the exclusion of others, and nothing in the character of the anæsthesia enables us to foretell what means will be effectual. Moreover the patients may vary in their idiosyncrasy to the influence of different agents from one moment to another.

(1) Two different metals applied simultaneously can reciprocally annihilate each other's influence. Vigouroux (61) says, "If in an hemianæsthetic patient, in whom the application of gold causes the anæsthesia rapidly to disappear, a piece of gold is applied, and above it a piece of silver, so long as they remain in contact the anæsthesia is not modified. If the silver is removed the anæsthesia disappears. If when sensation has been recovered, one does no more, it soon disappears and anæsthesia returns. But if the piece of silver is replaced, the sensibility remains so long as the silver is left there. The action of the silver has been in each case to render permanent the condition of things existing at the time it was applied." In an hemianæsthetic patient the phenomenon of transference, effected by an active metal, can be made to persist indefinitely by the application of a neutral one to the side formerly sensitive.

(2) When hysterical patients are improving, but have not regained complete health, before the anæsthesia completely disappears, it is in a sort of unstable equilibrium. If in this state an active metal is applied the anæsthesia reappears in all its original severity.

Theories advanced to explain these phenomena :—

(1) Physical theories.—The committee of the Société de Biologie with the aid of M. Regnard, came to the following conclusions (i.) Metals applied to the human skin determine currents of a sufficient intensity to be measured by a very delicate galvanometer; (ii.) the intensity of the current varies with the different metals and with the state of the skin; (iii.) the passage through the

skin of a galvanic current, of an equal intensity to the current determined in it by the application of an active metal, produces exactly the same effects on sensation as the metal itself.

They found further that a feeble current (*a*), or a strong current (*c*), caused a rapid return of sensibility while one of intermediate strength (*b*) had no effect. The difference in the action of the metals may be explained by supposing that in a certain patient gold produced a current of equal intensity to (*a*), iron to (*b*) and copper to (*c*); without invoking the aid of any mysterious agencies, gold and copper would then be active, and iron inert.

(*b*) On the other hand M. Vigouroux thinks that "the action of the metals is a phenomenon of static electricity." He brings forward several experiments in support of this, especially the fact of the neutralisation of the æsthesiogenic action of two metals by the superposition of one upon the other, which can hardly be explained on the supposition of electrical currents. Briefly he holds (1) that a certain electrical state of the peripheral organs of general and special sense is necessary for the performance of their functions. (2) This electrical state can be modified and with it the sensibility by producing on the surface of the body a change of tension, and this can be done by the application of a polarised plaque, by electrisation with one pole alone or by the galvanic current.

(*c*) Professor Schiff for several reasons does not believe in these electrical theories. He thinks with the physicists that material bodies are the seat of rapid molecular vibrations which they can communicate to neighbouring bodies. The variety of action of the metals will depend, according to him, on the number and rhythm of the vibrations of the æsthesiogenic agent, and of their concordance or otherwise with those of the nervous system of the subject.

According to another theory, supported by Hughes Bennett and Hack Tuke, the æsthesiogenic influence of these substances is to be referred to psychical causes, and is chiefly brought about by the imagination.

M. Pitres thinks that the phenomena of æsthesiogenesis do not all depend upon the same cause. The sensibility can be modified both by psychical and by physical influences. This we should expect, for there are many physiological and pathological phenomena which undergo variations dependent both upon external and upon psychical excitations. The proofs which demonstrate the intervention of physical causes in the production of certain of the æsthesiogenic phenomena are numerous and convincing.

On the other hand, the powerful æsthesiogenic influences of certain psychical factors—imagination, expectancy, moral emotions, and verbal suggestions is beyond question.

From the characters of hysterical anæsthesia, M. Pitres thinks that the localisation of the disorder of the nervous centres is in the basal ganglia, and that it is due to a functional inertia of these bodies. Anatomo-clinical observations show that destructive lesions, limited to the sensory region of the bulb or of the cerebral peduncles, give rise to anæsthesias that present all the clinical features of those met with in hysteria, except their variability. Experimental ablation of the higher parts of the brain affords support to the view, that the bulb may be considered as the nervous centre "for the change of impressions into sensations." From the fact that organic lesions of the posterior part of the internal capsule produce hemianæsthesia of the opposite side of the body, it has been held that hysterical hemianæsthesia depends upon a functional defect of these fibres. He adduces several observations in which, after hemiplegia of organic origin, there has been hemianæsthesia, which has undergone "transference," or has disappeared under the action of æsthesiogenic agents, and he thinks that this hemianæsthesia is not to be attributed to disease of the internal capsule, but to the indirect effects of the lesion in inhibiting the grey matter of the subjacent basal centres.

On the ground of the clinical differences between anæsthesias of cortical origin in organic disease and hysterical anæsthesias, Prof. Pitres cannot accept M. Binet's conclusions, based on experiments on hysterical subjects, that the lesion in hysteria, of whatever nature it may be, has its seat in the cortex.

As to diagnosis, three morbid conditions are considered to give rise to hemianæsthesia, with corresponding unilateral affection of the special sense organs; (1) a lesion of the posterior part of the internal capsule; (2) the intoxications, especially alcoholism and plumbism; and (3) hysteria. When such symptoms occur in the second group of cases, M. Charcot has shown that the patients are affected with hysteria, in addition to lead or alcohol (62). He also states, (63) "that hemianæsthesia due to organic lesion of the internal capsule, shows no essential difference from that of hysteria." The chief aid in diagnosis is that when, as is the rule, hemianæsthesia from an organic lesion is accompanied with hemiplegia, the lower third of the face is paralysed, whilst in hysterical hemiplegia, the face almost invariably escapes. Until 1889, M. Charcot's researches led him to believe that the anæs-

thesias disposed in geometrical areas, altogether independent of the nerve supply of the part—" en manchon, en gigot, en brassard " were typical of hysteria, but since that time observations on syringomyelia have shown that in this disease, the anæsthetic areas may have a similar arrangement. Moreover, the peculiar dissociation of the different forms of sensation characteristic of syringomyelia may be simulated by hysteria.

Hysterical Amblyopia and Achromatopsia.—Briquet observed that when amaurosis is unilateral, the patients are only occasionally aware of it.

Anæsthesia more commonly affects the eyes than the other special sense organs. Insensibility of the conjunctivæ, especially of the left conjunctiva, is so constant that it may be regarded as a characteristic sign of hysteria. When anæsthesia is complete, the oculo-palpebral reflex is absent. The loss of sensibility is superimposed on amblyopia. M. Féré (64) says: " The hysterical patients with hemianæsthesia that we have observed, and in whom there was neither contraction of the visual field, nor achromatopsia, preserved the sensibility of the conjunctivæ: those who have loss of vision for one or several colours, and have a more or less regularly proportioned contraction of the visual field, have lost the conjunctival sensibility, and those who have complete loss of colour vision, and almost complete loss of the visual field (for white), have lost sensibility over both conjunctiva and cornea."

Contact with the conjunctiva produces a flow of tears, even in cases where the oculo-palpebral reflex is lost.

The field of vision in hysteria is concentrically contracted. It is well-known that in the course of one examination, the field of vision in hysterical patients may be frequently modified, if the examination be too protracted, or so long continued as to fatigue the patient. The investigation must, therefore, be rapidly made, the determination of the field in its vertical and horizontal diameters being in most cases sufficient.

The contraction of the visual field is generally bilateral, but greater on one side, that of the hemianæsthesia, than on the other. Occasionally it is unilateral when it is on the side of the hemianæsthesia.

When the macula is invaded there is complete amaurosis. Unilateral is, relatively to bilateral amaurosis, the more frequent; the latter seems to be very rare. In the cases that M. Gilles de la Tourette has seen, the blindness was only

temporary in the great majority: it most frequently appeared after a convulsive attack, disappeared as suddenly as it came on, lasting some hours, or at most a few days. In one case, however, reported by Harlan (65) blindness persisted for ten years.

In hysteria, the different circles which correspond in exploration to the limits of the field of vision for each colour are, more or less, concentrically contracted, following the order recognised in the normal state. Vision for violet, green, red, and orange may disappear in the order named, these being the colours which are normally perceived centrally; the fields for the two first being the most restricted. Yellow and blue, the fields which have the greatest peripheral extent, disappear the last. Vision for these two colours is, therefore, longest preserved in hysterical amblyopia. But there are exceptions to this rule, red and not blue being frequently the last to go. In a still higher degree of hysterical achromatopsia the form of objects is perceived, but they all appear grey.

These disturbances of colour-sense are most marked in the eye on the anæsthetic side of the body, but the other eye generally shows them in less degree.

Permanent dyschromatopsia may suddenly give place to complete achromatopsia, under the influence of an impending convulsive attack; and this may disappear as suddenly after the paroxysm is over.

Central scotoma is rarely observed in hysteria. M. Parinaud (66) has found that central scotoma and peripheral contraction of the field of vision may develop simultaneously, so that perception of colours may be lost at the centre and at the periphery of the retina, and be retained in an intermediate zone which forms a ring surrounding the fixation point. Another peculiarity is that when anæsthesia to light and the contraction of the field for white are the most marked features, the field for red may be more extensive than that for white.

Acuteness of vision is as a rule very little modified in hysterical amblyopia, thus affording a remarkable contrast with the amblyopias that occur in atrophies of the optic disc.

Hysterical amblyopia alters very little the perception of form, but is often accompanied by other troubles of accommodation or of refraction such as the polyopia or diplopia that has been so well described by M. Parinaud (67). This polyopia is often overlooked, because it is generally obscured by amblyopia, and is only noticed by the patient when methodicially sought for. M. Parinaud gives the following rules for its detection (68). "A

pencil or some similar object is held before the eye and slowly removed. At first it is seen single; at 10—15cm. distance from the eye, a second image appears, generally on the temporal side. As the object is removed, the two images separate, and not seldom a third appears, less distinct, and on the opposite side. If the object is still further removed, the images become indistinct. At the same time the presence of macropsia or micropsia is shown, by the removal or approximation of the object."

No ophthalmoscopic changes have been found in cases of hysterical amblyopia.

M. Parinaud has observed that a certain number of patients cannot distinguish colours with either eye alone, and yet distinguish them very well in binocular vision. Also that in many cases of unilateral concentric contraction of the visual field, it is sufficient to open the sound eye for the field of vision of the amblyopic eye to increase from 10 to 20 degrees.

Monocular amblyopia, and even binocular retraction of the visual field does not inconvenience the patient much, so long as the acuteness of vision and perception of form is not modified. Similarly for achromatopsia; but one must bear in mind that railway servants who have developed achromatopsia as one of the symptoms of hysteria evoked by a collision may become incapable of carrying on their daily work.

How are we then to explain the fact that hysterical patients seem to suffer no inconvenience from their amblyopia in ordinary binocular vision? They cannot voluntarily neglect the image received by the affected eye, as persons who squint do, for they do not fix an object with the sound eye only, but place it exactly in the (principal) axis of binocular vision. M. Pitres believes that the amblyopia is corrected, because it exists only in monocular vision and disappears at once when both eyes are used. He tested a patient, who had barely perception of light with the left eye, with Flees' box, an apparatus in which two points of different colours are seen, one to the right and the other to the left, and by an ingenious arrangement the point to the left is seen with the right eye and *vice versâ;* he also employed the prism and the screen tests. To all these tests she answered correctly, showing apparently that binocular vision was normal. He quotes some observations of M. Parinaud, who found that certain patients had achromatopsia for each eye in monocular vision, and yet distinguished colours perfectly in binocular vision. Two explanations of these facts have been proposed, the first, sup-

ported by Ferrier's experiments and which the author considers most probably correct, that the cortical centres for binocular and monocular vision are distinct, and that the latter only are affected in hysterical amblyopia. The second, advanced by M. Bernheim, that hysterical amblyopia (*cécité*) is a purely psychical phenomenon, an illusion of the mind. "The hysterical person," he says, "neutralises unconsciously by the aid of his imagination, the visual image that is normally perceived." The author forcibly refutes the argument that hysterical amblyopic affections are simulated.

Causes of amblyopia.—The onset is generally insidious, but a local traumatism (to the eye) may according to the law established by Charcot, develop local hysterical phenomena. The prognosis is good.

Differential diagnosis.—Lesions of the posterior third of the internal capsule, according to M. Charcot, are accompanied by hemianæsthesia with concentric contraction of the visual field and dychromatopsia identical with that observed in hysteria; this contraction of the visual field is bilateral, and M. Charcot has therefore rejected the term crossed amblyopia by which he at first designated this affection.

The amblyopia of lead-poisoning belongs in reality to hysteria, as do many cases of alcoholic amblyopia also. Cases of genuine alcoholic amblyopia also occur, and are distinguished by the presence of a central scotoma spreading to the periphery (69). It is true that central scotoma may occasionally occur in hysteria, and a case of combined hysteria and alcoholism has been reported by M. Charcot (70) in which there was both concentric contraction of the field (hysteria) and central scotoma (alcohol), but a knowledge of these variations will be sufficient to prevent mistakes.

Disseminated sclerosis is frequently associated with hysteria, and syphilitic lesions have been found in the fundus of an eye affected with hysterical amaurosis. MM. Déjèrine and Tuilant (71) have stated that in syringomyelia a concentric contraction of the visual field for colours is found, but only corresponds to that of hysteria in the field for white. For other colours the visual field is irregular, notched, and shows the greatest contraction for green; all these features being unlike the phenomena of hysteria, and more resembling those found in atrophy of the optic disc.

Opthalmoscopic examination showed no lesion in the se cases. As no other examples of the same kind have been reported, whilst many cases of syringomyelia have been published, in which, apart from a morbid association of the disease with hysteria, the visual

fields have been normal, the author concludes with M. Brianceau (72) that—

(i.) In the majority of cases of syringomyelia the visual field remains normal: contraction of it is not a symptom of the disease.

(ii.) When it is retracted an explanation other than the cord lesion is to be sought for.

(iii.) Hysteria, so often associated with syringomyelia, is the only cause of contraction of the visual field in this disease apart from local ocular affections.

M. Féré (73) states that all the manifestations of epilepsy attended with loss of consciousness are followed by amblyopia for a certain time: he always found a decided contraction of the visual field (18 patients), when he examined the patients within half-an-hour after consciousness had been regained. This contraction is accompanied by a diminution of the acuteness of vision by two, three, or four-fifths. The sensibility to light and to colours is also lessened (after the fits) and this diminution seems to be the counterpart of the hyper-sensibility to light and to colours—particularly red (Hammond)—that is sometimes observed in the prodromal period. Further M. Féré has three times found anæsthesia of the eyelids, conjunctiva, and the circumferential skin around the eye coincidently with complex visual troubles and nystagmus. He does not admit the existence of permanent contraction of the field of vision except in cases where the attacks closely follow each other. MM. Oppenheim and Thomson on the other hand, in 94 cases found in only 4 temporary, while one-third of the cases showed permanent contraction of the visual field (74). M. Pichon (75) similarly found a permanent contraction of the field of vision in one-third of his patients, but he insists on its irregularity of contour. M. Hitier however, investigating the question anew on 87 epileptics in the service of M. Charcot, found a permanent contraction in 3 only. M. Charcot teaches that permanent contraction of the visual field in epilepsy is "une quantité négligeable," above all when it is compared with the temporary contraction that follows the fits. In the great majority of cases he says some contraction follows the fits, but it may occasionally precede them as the aura. When however the fits follow each other at short intervals, every 6 or 8 days for instance, the contraction of the visual field is to some extent a permanent symptom.

Hysterical affections of the ocular muscles.—These affections in the great majority of cases accompany amblyopia, and often for a short time relatively to the persistence of amblyopia in hysterical patients. Sometimes—and this is especially true of one

variety, blepharospasm—they exist alone, as the sole symptoms of an "abortive" hysteria, and the most frequent instances of this, so far as regards the localization of the symptoms in the eyes, are found in children. Most often however they are accompanied with other well-marked signs of hysteria.

There may be loss of the muscular sense in the orbital muscles. Szokalski (76) says, "If the anæsthesia of the ocular muscles remains free from all complication with retinal anæsthesia, the faculty of vision is disturbed by loss of the muscular sense in these muscles, or loss of the sense of position of objects seen. The patient seems to lose the perception of height and depth, of what is vertical and what is horizontal, &c."

M. Borel (77) has undertaken some experiments to elucidate this question. The subjects were a man and a woman suffering from total anæsthesia, with absolute loss of the muscular sense in all four limbs. The man (who fell like a log as soon as his eyelids were closed), with one eye shut was made to look through a tube so directed as not to permit of his seeing any object which was familiar to him and could help him. The outlook from the tube was closed at some distance by the hand. One tried to make him follow with the eye alterations in the axis of the tube, and after noting the position of the eye, he was questioned as to its direction. He always gave wrong answers, when, indeed, he did not indicate that he had no idea of the direction of his eyes. He did not know whether he was looking up or down, to the right or to the left. It sufficed for the tube to be well fitted to the globe of the eye so that no familiar object could be seen to put him completely at fault. The same procedure gave identical results in the other patient. Loss of muscular sense may accordingly be as complete in the muscles of the eye as in those of the limbs.

Monocular polyopia and micro-megalopsia appear to be caused by a contracture of the ciliary muscle. M. Parinaud says that the ciliary muscle is very often affected in hysteria. Its contraction generally accompanies that of other muscles of the eyeball and of the orbicularis, but may exist alone. It is often overlooked as it is masked by the amblyopia present. This contraction of the muscle of accommodation produces effects analogous to those of myopia, that is to say the p.r. is more or less approximated. But hysterical contracture differs from myopia in that the muscle being immobilised in the position of contraction, the range of accommodation is nil, the p.r. and p.p. are the same, and the eye is adapted for one distance only. This contraction may be

extreme and the point of distinct vision be very near the eye. In myopia the same degree of contraction will bring this point nearer, in hypermetropia place it further away. In the last case the eye being adapted for a considerable distance convex glasses will correct the vision for near objects, *e.g.*, for reading. In cases of this kind the condition may be mistaken for paralysis of accommodation which is a very rare phenomenon in hysteria. M. Parinaud explains monocular polyopia in the following way. The crystalline lens is not completely homogeneous in structure. The segmentation of its substance together with the modifications of curvature that it undergoes in accommodation render it comparable to a lens divided into several segments, which are re-united, but not fused together, in such a way that the images formed by each of the segments are combined at the common focus but remain distinct on the near and far side of it. The normal action of accommodation, which is to bring the rays of light always to a focus on the retina, hides this defect of structure, but it becomes manifest in certain troubles of accommodation. In this way atropine produces for certain distances monocular polyopia. It fixes the ciliary muscle and brings out especially in young subjects the segmentary state of the lens whilst at the same time exaggerating its curvature. M. Parinaud attributes micro-megalopsia to a similar contraction of the muscle of accommodation. It is more frequent than monocular polyopia, which arises from the same cause, and which requires for its production, a stronger contraction and certain peculiar conditions.

Blepharospasm is the most frequent contraction of the ocular muscles. It may occur in hysterical subjects from very slight causes. Numerous instances have been reported which place the influence of hysterogenic or hyperæsthetic zones on the production of blepharospasm beyond dispute. Hysterical blepharospasm may be clonic, tonic, or take the form that has been called by M. Parinaud "pseudo-paralytic ptosis." The clonic form is the most common and also the most transitory. It is always bilateral. Tonic blepharospasm occurs in two forms, painful and not painful. In the former it is as a rule bilateral, and the muscular contraction is so forcible that it can only be overcome with difficulty or not at all. There is generally photo-phobia and lachrymation. In the latter the affection is often unilateral. The orbicularis is actively contracted, the upper eyelid thrown into folds, and the patient's efforts to open the eye only result in feeble contractions of the muscle, which accentuate the closure of the lids, whilst the upper eyelid shows from time to time convulsive movements or flick-

erings. When one tries to raise the eyelid a very appreciable resistance is felt. In the pseudo-paralytic form, the tonic spasm is so to speak absent; when the patient is told to open the eye he instinctively throws the head backwards and the frontalis contracts energetically, as in true paralytic ptosis. But the upper lid covers the lower exactly, when it is raised with the finger it descends again more energetically than in paralysis, and the convulsive flickerings of the upper lid may be seen in this form also. M. Charcot has recently (78) pointed out that the eyebrow is lowered in spasm whilst in paralysis it lies higher than that of the healthy side. Lastly M. Gilles de la Tourette has discovered a symptom which he regards as pathognomonic, and which consists in the addition of sensory troubles to the spasm. In the painless form of blepharospasm there is a zone of anæsthesia circular in shape covering the eye and the surrounding skin for a distance of 2cm. beyond the orbit. The conjunctiva, palpebral and ocular, is almost completely insensible. On the cornea sensation is preserved over its internal segment only.

In painful blepharospasm there is the same circular zone, but in this case of hyperæsthesia, instead of anæsthesia. These phenomena are analogous to the sensory troubles "in geometrical-territories" superimposed on loss of function, and independent of the distribution of the peripheral nerves, that MM. Charcot and Féré had already noticed in cases of hysterical amblyopia.

Blepharospasm is almost always associated with amblyopia. It is generally of short duration, but may last some years; like other hysterical phenomena it may disappear suddenly. The surgical procedures which have been advocated, namely, stretching, excision of the nerve, or section of the orbicularis muscle are quite unjustifiable.

M. Parinaud (79) states that tonic hysterical blepharospasm is almost always accompanied by contraction of some of the ocular muscles, but that isolated contraction, at least a state of fixed contraction, is rare in these muscles, though a passing diplopia is common enough. Spasmodic strabismus in hysteria has resulted from the presence of carious teeth or alveolar periostitis. During the convulsive attack, at the period of the tonic or clonic convulsions, contractions of the ocular muscles may be noted, and they may sometimes coincide with contractions of the neck muscles, so that conjugate deviation of the eyes and head may be simulated, and suggest an organic cerebral lesion. Generally, however, the above syndrome is not so accentuated, the eye is carried in all directions by rapid movements of short duration without

any regularity; at the same time the pupils are agitated by spasmodic contractions and dilatations. Permanent deviation of the eyes is much more rare. A case is recorded by Frost (80), in which there was conjugate deviation of the eyes downwards and to the right. The author considers that in almost all, if not all, the reported cases of hysterical paralysis of the third nerve, the accuracy of the diagnosis may be doubted. He cannot at all accept Schæfer's conclusions (81) on hysteria in children, that whilst paralysis of the facial and hypo-glossal nerves is rare, that of the third nerve, or isolated paralysis of the branch to the levator palpebræ is sufficiently common. The only case of hysterical paralysis of the third nerve that is above dispute, is one communicated by Parinaud and P. Richer, and even that dates from ten years ago, when the diagnosis between spasm and paralysis was not so well differentiated as it is to-day. In this case there was paralytic ptosis, paralysis of accommodation, and incomplete palsy of the right internal and inferior rectus.

Permanent dilatation or contraction of the pupils is very rare in hysteria, and is generally observed as a transitory phenomenon of the convulsive attacks. In some cases dilatation of the pupil has been noted following myosis due to spasmodic contraction of several days' duration, and may be compared to the weakness of a limb which follows a state of hysterical contraction. Spasmodic mydriasis has been described by Giraud Teulon (82) and Duboys (83).

Insufficiency of convergence, from weakness of the internal recti, is so bound up with other ocular motor troubles, that it will only very rarely be met with as an isolated symptom.

As to the question of hysterical paralysis of the sixth nerve, M. G. de la Tourette quotes three cases only, one of which was a case of associated paralysis of the sixth pair of nerves in looking to the left or to the right; in the two others the paralysis at first affected the external rectus of one eye, and then passed to the same muscle of the other eye. This alternation of the paralysis appears to be typical of hysteria, the paralysis of organic origin being much more fixed, and the secondary spasm never showing the same degree of intensity.

Cases of external ophthalmoplegia in hysteria have been reported by Ballet (84), Bristowe (85), Parinaud (86), and Raymond (87). These observations show that external opthalmoplegia in hysteria has some peculiar characters which differentiate it from the same symptom when due to an organic lesion. There is an entire absence of all voluntary movement, while unconscious (in-

voluntary) movements are preserved: "the eyes are not constantly fixed, but execute movements of limited extent in different directions." There is no mydriasis; further the ophthalmoplegia remains external, and is not internal or total.

Hysterical hyperæsthesia.—Hyperæsthesia may affect the skin, mucous membranes, or viscera.

In searching for hyperæsthesia, it is important to examine a sufficiently extensive surface at a time. Further, stroking the skin with the finger will be much more vividly felt than a prick, especially a deep prick, for the skin itself and not the underlying tissues is the seat of the pain. Cutaneous hyperæsthesia is especially found in the hysteria of adults, male and female. It appears and disappears spontaneously. Convulsive attacks have a marked influence, hyperæsthesia appears or disappears after them, or is replaced by anæsthesia, and *vice versâ*.

In distribution, hyperæsthesia is rarely general. M. Gilles de la Tourette has observed a few cases of this in men. Most commonly it is disposed in patches. Unlike anæsthesia there is no need to search for hyperæsthesia, the patient is acutely conscious of it, partly from exaggeration of subjective sensations (numbness, formication, prickings, tinglings), and partly from exaggeration of sensations excited directly by external agencies (blows, rubbing, &c.).

Occasionally hyperæsthesia is unilateral. In one case described by M. Charcot, there was hyperæsthesia on one side of the body, whilst the other side was anæsthetic. Sometimes hyperæsthesia occurs in geometrical areas; it is then superimposed on the loss of function of the part, *e.g.*, in a case of contraction of the orbicularis palpebrarum, there was a zone of hyperæsthesia exactly overlying this muscle. Hyperæsthesia probably appears first, and always persists a certain time after the contraction has gone.

Hysterical Joint Affections.—According to M. Charcot, the knee is the joint most often affected. Of seventy cases the knee was affected thirty-eight times, the hip eighteen times, the wrist eight, the shoulder four, and the ankle twice.

Causes (1) Injury, a fall, blow, or unaccustomed fatigue, such as a prolonged march. M. Charcot has shown that to have their due effect, these traumatisms, which are often insignificant in themselves, must take place in persons whose mental state is peculiar, such as hysterical persons who at times are as open to suggestion as those who are in the hypnotic state. There is a "traumatic suggestion" rather than an actual traumatism, for

there is always, between the reception of the injury and the manifestion of the hysterical symptom, a period of preparation, "méditation psychique." Imitation, as Paget has shown, sometimes plays an important part. The age of the patients varies. Men are frequently affected. In opposition to Briquet M. Charcot teaches that an hysterical joint may be one of the first manifestations of the neurosis.

The first and earliest symptom of an hysterical joint is pain, the second, spasmodic contraction of the muscles which put the joint into action. This contraction may spread to all the muscles of the limb. The third is loss of power in the limb. Brodie's classical description of the symptoms is quoted, in which it is pointed out that the seat of the pain is in the skin. In the case of the hip the area of cutaneous hyperæsthesia is roughly triangular, the apex of the triangle being at the root of the scrotum, or mons Veneris, and the base running parallel to the fold of the groin, the triangle enlarges progressively as it turns round the buttock to pass towards the sacrum and encroaches on the hypogastric region. In the knee the hyperæsthesia extends round the limb a hand's breath above and below the middle of the joint, it has a similar distribution for the ankle and the joints of the upper extremity. This hyperæsthesia is added to the loss of function of the joint, and its distribution may be compared with that of the anæsthesia which accompanies spasmodic contractions of the orbicularis palpebrarum, or of the limb muscles. These hyperæsthesias or anæsthesias of geometrical distribution appear before the loss of function and persist after it. Sometimes these hyperæsthetic areas become true hysterogenic zones.

Charcot (88) and Babinsky (89) have shown that muscular atrophy analogous to that described by Valtat (90) and accompanied by the reaction of degeneration and fibrillar trembling may be present in cases of hysteria arthralgia. This muscular atrophy is less precisely localized than in cases of organic disease, in which it always affects the extensors of the joint: moreover it is much more rare. Examination of the joint under chloroform should be practised. M. Charcot (91) has shown that the order in which the painful phenomena return with the regain of consciousness after chloroform, is of diagnostic importance. He says "During the return to consciousness, rigidity began to reappear to a certain degree in the affected muscles before any manifestation of pain in the joint occurred. The sensibility of the skin had already partly returned, the patient had begun to reply to some questions, at a time when the sensibility of the

deeper parts (percussion of the trochanter or heel) was still in no way exaggerated."

In a case of organic hip-disease pain returns first in the deeper, afterwards in the superficial parts. The changes that have been found in the joints and bones in long-standing hysterical cases can be accounted for by the want of use of the parts. The duration of hysterical arthralgia may be months or years, the general course being interrupted by temporary recoveries, to be again followed by a relapse.

M. Charcot has shown that when anæsthesia or hyperæsthesia of geometrical distribution exists in addition to a contraction or paralysis with or without arthralgia, the sensory disturbance always persists some time after the disappearance of the functional troubles. So long as the sensory disturbance persists one ought to fear the return of the paralysis, contraction, or arthralgia. Like all other hysterical affections arthralgia may undergo a sudden cure.

As a rule, the cause of the appearance of hyperæsthetic zones remains unfathomable. In a case reported by M. Charcot, however, a man fell into the water whilst fishing, and the cord of the net became entangled round his left leg. He was nearly drowned, and a patch of hyperæsthesia was afterwards found over the spot compressed by the cord. M. Pitres also relates the case of a patient "who imagined that she was, from time to time, visited by an old woman who touched her in different parts of the body, and on all the spots thus touched, there was afterwards hyperalgesia which persisted for some days."

Hysterogenic zones.—Hysterogenic zones are circumscribed regions of the body, which may or may not be painful, from which there often pass, during the prodromal stage of the spontaneous attacks, special sensations which play a part in the whole of the phenomena of the hysterical aura. Compression of these spots has for its effect, either to determine a convulsive seizure, or a part of the spasmodic phenomena of the seizure, or to arrest suddenly convulsions already begun. This compression not only arrests the convulsive attacks, but also attacks of rhythmical chorea, of cough, hiccough, &c., in short, every kind of paroxysm. Hysterogenic zones are excitant (spasmogénes) or inhibitory, (frénatrices) of convulsive attacks, some, especially those which are not the seat of hyperæsthesia, are "spasmo-frénatrices," that is to say, light pressure may provoke and firm pressure arrest an attack. These zones are especially found in hysteria with convulsions. They are often

of small dimensions, of the size of a two or five franc piece. The skin over them is generally anæsthetic, a fact which differentiates them from areas of simple hyperæsthesia, it may, however, be hyperæsthetic. When seated on the hemianæsthetic side of a patient they may easily be overlooked. Firm pressure is necessary for their discovery. The occurrence of spontaneous pains may be a useful guide to the position of hysterogenic zones. They appear and disappear most frequently under the influence of an hysterical fit.

MM. Bourneville, Regnard, and P. Richer state that hysterogenic zones occur only on the head and trunk, but M. Pitres has also found them on the limbs. According to him, the zones on the limbs and thorax are exclusively "excitant" (spasmogénes), and those over the ovarian and epigastric regions, "inhibitory" (frénatrices).

He classifies hysterogenic zones according to their anatomical distribution into—

(1) Cutaneous, which are the least common.

(2) Subcutaneous, which are more frequently met with. In this variety, convulsive attacks are determined by pressure on subcutaneous tissues, irritation of the skin, or of the underlying muscles and tendons being without effect. He thinks that the seat of the irritation is in the nerves, and that the nerve tracks leading to the zone, if compressed in any part of their course between the spinal cord and the zone, can equally well excite an attack. This statement applies to the subcutaneous zones only.

(3) Visceral zones are situated in the parenchyma of complex organs, such as the mammary gland, ovary, and testicle. In such cases, the skin overlying the affected organ has no hysterogenic properties.

On some special hyperæsthetic hysterogenic zones.—Those on the scalp are some of the most important and may be single or multiple; when single, they are generally situated at the vertex. They are of limited extent and the sensibility over them is exquisitely acute; spontaneous radiations of pain start from them. Hysterical cephalalgia is found in six-sevenths of hysterical female patients according to Briquet, but though often caused by the presence of an hyperæsthetic or hysterogenic zone on the scalp, this is not always the case. Briquet concludes his description of "clavus" thus. "The pain of clavus is fixed and does not move about. It lasts several days or even three or four weeks. It is frequently accompanied by attacks of shivering, vomiting,

digestive disturbance and sometimes by fever." This observation contains the germ of the description of a recently studied group of symptoms, namely, hysterical pseudo-meningitis. In this affection the symptoms of meningitis are very closely simulated. It generally occurs in adult women but has been once observed in a little girl of five (92). The mode of onset may be sudden or preceded by a prodromal period of malaise as in tubercular meningitis. The chief symptom in the latter case is headache which shows itself in the form of acute exacerbations and coincides with the presence of hysterogenic or hyperæsthetic zones on the scalp, which should always be sought for. These exacerbations are accompanied by cries and groans. Sometimes there is delirium. There is often photophobia and diplopia from passing strabismus: there may be rigidity of the neck muscles, opisthotonus, and contractions of the arms or of the legs. The pulse may be slowed, but according to Pitres is never irregular. Hyperæsthesia and tâche cérébrale are present, and constipation and vomiting complete the list of symptoms, which thus closely resembles those of tubercular meningitis. In one case only was the temperature raised to 39.2° (102.5 F.) in the others it did not exceed 38.6° (101.4 F.) and in several remained normal. Meanwhile the evolution of the symptoms is not continuous but proceeds by exacerbations or paroxysms. The presence of some concurrent and independent acute inflammatory affection must be looked for, since this may be the cause of the fever in cases where pyrexia is present; for instance, vaginitis was found in two cases. M. Chantemesse (93) says that the mode of onset and the progressive evolution of the symptoms cause them closely to resemble tubercular meningitis. Only the knowledge of the patient's antecedents, of the presence of sensory troubles attributable to hysteria and the normal temperature should make one suspect hysteria. He (94) has also made use of the observations of MM. Gilles de la Tourette and Cathelineau on the changes in the urine in hysterical paroxysms. They showed that during hysterical paroxysms the amount of urea excreted is diminished and the proportion of the earthy to the alkaline phosphates, which is 1-3, both in the healthy and in the hysterical subject apart from the paroxysms, becomes 1 to 2 or equal. M. Chantemesse finds that these changes are found in the urine of persons suffering from hysterical pseudo-meningitis, and this disorder must therefore be probably regarded as an attack or crisis spread over a long period.

M. Gilles de la Tourette has observed five cases of facial

neuralgia of hysterical origin. In one patient in Prof. Charcot's clinic, there had occurred three attacks of facial neuralgia at different times. An hysterogenic zone was found at the point of exit of the nerves, and another of the same kind in the intercostal region. Pressure on the zones excited an attack of neuralgia ending in a convulsive attack. These neuralgias are to be considered as a painful prolonged crisis when they end in a fit of convulsions, and as a complete hysterical paroxysm when no convulsions occur. The radiation of the pains is interesting: it not rarely occurs between neighbouring hysterogenic zones; for instance the pains may start from the vertex and arouse hysterogenic zones of the fifth pair, or acting at a still greater distance compression of an ovarian zone may excite an attack of hysterical migraine. As to diagnosis from true facial neuralgia M. Charcot lays stress on the occurrence of the hysterical affection in the evening, whilst the genuine attack generally takes place in the morning. Most important is the co-existence of other hysterical stigmata. The author thinks that when the nature of a facial neuralgia is uncertain, an estimation of the urinary solids during the period of 24 hours in which the attack occurs should be made and compared with that of another period of 24 hours free from the paroxysm.

Rachialgia is one of the most common hysterical manifestations.

The hyperæsthetic hysterogenic area may extend the whole length of the spinal column, more often it stretches only over four or five vertebræ, generally in the dorsal and lumbar region. It is particularly well-marked over the spines and does not pass laterally beyond the spinal furrow. It is tender to the slightest touch and is generally the seat of spontaneous pain. Pressure upon the zone may determine a convulsive attack. The observations of Brodie on rachialgia are complete and well known. It should be noted that M. Paul Richer (95) has pointed out that the lumbar spines are, in some persons, especially when they bend forward, very prominent, and that this normal prominence has been frequently treated as pathological.

Sometimes rachialgia is accompanied by a lateral hyperæsthetic or hysterogenous zone on the trunk,—pleuralgia of Briquet—it extends round one side of the body from the spinal column behind to the middle line in front, and is generally from 4-5 fingers' breadths in width and seated about the level of the 5th, 6th, 7th and 8th intercostal spaces. The pain is aggravated by breathing, coughing, and walking.

The knowledge of the occurrence of this hyperæsthetic zone is important, for with a painful area over the dorsal spine, spontaneous pains, and sensation of constriction round the waist, the girdle-pains and exquisite sensibility of the thorax and spine found in some cases of locomotor ataxy may be closely simulated. The hyperæsthetic hysterogemic zone of the vertebral column may be localised in its inferior extremity when it constitutes hysterical sacrodynia and is a very obstinate affection. Its nature can be recognised by the co-existence of other hysterical stigmata, and by the extreme sensibility of the overlying skin, in which the pain is chiefly localised.

In men two hysterogenic cutaneous zones are often found. One pseudo-ovarian is seated in the region which corresponds to the ovary in the woman. It is generally unilateral and on the anæsthetic or hyperæsthetic side of the body, but may be bilateral. Its extent is limited and the skin over it is often insensible, deep pressure being necessary to induce an attack. The second is seated in the skin of the scrotum (not in the testicle itself), and it is almost always unilateral.

Hysterogenic zones on the mucous membranes.—Our knowledge of these zones we owe largely to the observations of a pupil of Professor Pitres, M. Lichtwitz. M. Lichtwitz has found only spasmogenic zones, apart from the "hypnogenic" zones to be mentioned later. They are situated on the mucous membrane of the nose, mouth, upper air passages, external auditory meatus and lachrymal canal. They frequently co-exist with cutaneous hysterogenic zones in the same subject. He found that they occurred more particularly on the anæsthetic side of the body, a statement which M. Gilles de la Tourette thinks requires further investigation. Unlike the skin, which shows no naked-eye changes over the position of a zone, obvious morbid changes are frequently present in the affected mucous membranes: *e.g.*, inflammatory affections of the larynx and external auditory meatus, atrophy or hypertrophy of the mucosa over the lower turbinated bones. Beyond direct pressure these zones may be excited by faradic and galvanic currents, by chemical substances, and by inhalations. It is important to note that in some cases the cure of the local morbid changes in the mucous membrane has been followed by disappearance of the hysterogenic zones and of the hysterical symptoms. The special senses differ in the way in which they react to the various hysterical excitations. When an hysterogenic zone is situated on the lingual or nasal mucous membrane the trouble excited by it remains as a rule local, as a perversion of

smell. When the mucous membrane of the tympanum, middle ear or eye is implicated, more complex phenomena follow the irritation of the zone. The excitation of hysterogenic zones localised in certain regions may give rise to a symptom-complex generally observed under very different circumstances, for instance facial neuralgia, when the aura is seated on the branches of the fifth. In the case of the ear the effects influence the nerves of the labyrinth and convulsive attacks arising from a zone in this situation may exactly simulate Menière's disease. The diagnosis may be made by the characteristic changes in the relative proportion of the urinary solids already referred to as occurring during an hysterical convulsion.

Hysterogenic zones may be seated on the conjunctiva, cornea, or lachrymal duct, and irritation of them may give rise to a convulsive attack, in which all the symptoms of migraine may be simulated. The chief points in the diagnosis of the hysterical form are the following. Migraine may constitute the whole of an hysterical paroxysm, or be the starting point of a convulsive attack. Besides the ordinary prodromata of an hysterical attack, throbbing in the temples, globus, &c., there is almost always an hysterogenic zone which forms the starting point of the paroxysm; this zone may be seated in the eye itself, or on the vertex. In one case the pain began at the vertex, passed to the left supra-orbital region, and was followed by a luminous scotoma. In two other cases, pressure on an hysterogenic zone in one over the sixth dorsal vertebra, and in the other over the left ovary immediately caused the appearance of the scintillating scotoma (96). When migraine forms the aura of an attack, or when it constitutes the whole (specialised) paroxysm and starts from an hysterogenic zone, irritation of which determines the attack, the presence of the hysterical form of migraine is certain. In doubtful cases the author relies on an examination of the urine (see below). Hemiopia is often observed in ordinary migraine, and may be present in the hysterical form, as a temporary phenomenon of the attack, but is never permanent. In these latter cases there is the usual concentric contraction of the visual field, and M. Parinaud, examining an hysterical patient in whom an attack of migraine with hemiopia could be induced by hypnotic suggestion, found that, during the presence of hemiopia, the visual field was still more contracted than in the usual state, but the objective characters of permanent hemiopia were not present.

Vaginismus is most often the result of a spasmodic contraction of the vaginal muscles brought about by irritation of an hyperæsthetic or hysterogenic zone.

The viscera most frequently affected are the breast, the testicle, and the ovary. In the first two the zone is generally unilateral, and may be seated either only in the skin covering the testicle or breast, or in the glands themselves. Professor Pitres says that the painful affection described by surgeons under the name of irritable testicle, is sometimes the consequence of a symptomatic neuralgia of the spermatic plexus, but in a large number of cases appears to be due to a simple hysterical hyperalgesia of the parenchyma of the testicle. Castration has been practised, but the pains have often persisted after removal of the testicles, proving that the hyperalgesia is not always due to local irritation of the nerves of the spermatic plexus, and that it depends, at least sometimes, on a functiona modification of the sensory nervous centres.

Hyperæsthetic hysterogenic ovarian zone.—The efficacy of pressure on the abdomen in arresting hysterical convulsive seizures was known to Mercado in the sixteenth century; Schutzemberger exactly localised the spot in 1844; he says that the ovary forms the most frequent point of departure of an hysterical spasm. Briquet pursuing his general theory as to the seat of hysterical hyperæsthesia localised ovarian pain in the abdominal walls; he gave it the name of cœlialgia.

An ovarian zone rarely passes unnoticed for it is often the point of departure of particular sensations which attract the patient's attention. Ordinarily there is between the attacks a dull pain, brought out by walking or by the pressure of a corset. For some hours or days before a convulsive seizure, the pain becomes sharp, lancinating, and radiates into the epigastric region, to be followed by globus, in a word the common sensations of the classical aura happen. After the attack is over the pain becomes less acute. Sometimes it is sufficiently severe to cause the patients to walk bent forwards and inclined to one side in order to relax the abdominal muscles.

In some cases the skin is hyperæsthetic in a limited area over the site of the ovary. The author believes that these cases have been examined shortly after convulsive attacks, when the skin is itself the seat of an hyperæsthetic zone, for in the great majority there is on the contrary a very circumscribed zone of cutaneous anæsthesia lying above the ovary. According to M. Charcot's writings, by deep pressure at the intersection of the line joining the anterior superior iliac crests and that limiting the epigastric region laterally, the brim of the pelvis may be felt, and at about the middle of this rigid crest a small ovoid body will be felt to

glide under the fingers. Pressure on this brings out not ordinary sensations of pain but those of the hysterical aura. In two cases where the patients were pregnant, this ovarian zone was observed to rise with the normal ascent of the ovary during pregnancy. M. Guinon reports an additional instance in point. This zone is generally unilateral, it is one of the most fixed, active, and powerful zones; it generally has two properties, light pressure upon it exciting, strong pressure arresting, an hysterical attack.

Hysterogenic zones can be made to disappear by (1) general and (2) local means.

(1) Includes static electricity, the galvanic current applied to nervous centres, ether or chloroform anæsthesia.

(2) Artificial local anæmia, by Esmarch's elastic band round a limb, application of cold (ether spray), a mixture of ice and salt, of a mustard poultice, hypodermic injections of pure water, localised electralisation. The action of any of these measures is only temporary.

M. Pitres says that "in localised electrisation" of the zones, the current must be differently applied, according as subcutaneous or cutaneous zones are in question. In the first case, the electrodes may be applied directly over the skin of the hysterogenic region. In the second, the zone itself must be avoided. The electrodes must be placed beyond the limits of the excitable region. Further, the currents employed must not be too strong, or they may produce convulsions before the zone has lost its excitability.

The prodromal period of a spontaneous hysterical seizure is marked in its complete form by three stages. (1) The psychical aura, marked by ideas of sadness, terror, &c.; (2) hysterogenic aura, acute pains at situation of the hysterogenic zones; (3) ovarian aura, the sensation of a ball which rises upwards from the ovarian regions, and immediately preceeds the onset of convulsions. A convulsive attack may be artifically provoked by giving rise to the sensations of either of these three periods. M. Pitres thinks that possibly the clumsy handling of a patient in an hysterical seizure may give rise to irritation of hysterogenic zones and so prolong the attack. The knowledge of the presence of these zones is important to the physician for (1) he will take care that hysterical convulsions are not prolonged in the above way. (2) He will be able to artificially provoke an attack by their means, with the object of putting an end to long-continued painful auras, or of modifying certain permanent hysterical symptoms, *e.g.*, contractures, or of confirming a doubtful diagnosis. (3) He will be

furnished with a very efficacious means, in many cases, of arresting the attacks.

The provocation of a paroxysm may be as useful as its arrest in modifying the manifestations of hysteria actually present. For instance an hysterical paraplegia may be brought to an end by inducing a convulsive attack by the aid of an hysterogenic zone. The paraplegia may indeed be replaced by a spasmodic contraction or some other hysterical phenomenon, but an hysterical manifestation of recent date is always more easily relieved than one of long standing. The displacement of an hysterical phenomenon is already a step towards its disappearance. Certain hysterical states, *e.g.*, hysterical sleep which may last for days or weeks, may often be suddenly brought to an end by pressure on an hysterogenic zone.

Hysterical or pseudo-angina pectoris.—Hysterical angina, unlike the true form, occurs especially in those under 40. Whenever an attack has occurred, it is probable that other paroxysms in the same patient will take this form. Hysterical angina is a rare affection. The attacks are generally sudden and often nocturnal, sometimes they recur with a regular periodicity. There may be an hyperæsthetic or hysterogenic zone in the præcordal region, or over the mid-dorsal region of the spine. The attack begins by an increase of pain and tenderness in these zones. Sometimes the aura may begin with pains in the testicles or abdomen. The pains radiate to the neck and down the left arm to the fingers, or they may start from the fingers and pass to the chest. This is the neuralgic form. In the "vaso-motor" form the face becomes pale and cold, then livid, red and purplish, and the same changes may affect the left arm. The state of the heart, pulse, and respiration show the greatest differences in different cases. The pupils may be dilated or contracted. The patient may experience a feeling of inexpressible terror, or may have a tendency to syncope. A kind of dream of the attack may pass through all this period. In the diagnosis from true angina pectoris the exciting cause is important. Moral agencies, such as a contrariety, bring on the hysterical, while physical causes induce the organic form. The pulse and respiration generally remain calm in true angina—the cardiac erethism so especially related to the cries, groans, and agitation of the hysterical crisis does not occur in them.

The number of attacks is uncertain. One patient had more than two hundred in the course of two years. In diagnosis, the family and antecedent history of the patients, must be care-

fully enquired into, and the presence of hysterical stigmata sought for; the analysis of the urine on the lines mentioned above, will be of service; organic disease of the heart and great vessels must be excluded, the symptomatology of the attacks studied, and by this means an accurate conclusion can generally be arrived at.

The convulsive seizures of hysteria.—Hysterical convulsions are more common in men than in women, of M. Pitres' cases, 56 out of 61 women, whilst only 7 out of 31 men, had convulsions. A strong emotion is most often the cause of the first attack, afterwards they may arise spontaneously, or be excited by the most trivial causes.

To M. Charcot, and after him to Paul Richer, the credit is due of being the first to give a systematic description of hysterical convulsive attacks and to show that they may be divided into several phases, succeeding each other in regular order. The "grande attaeque" of hystéro-épilepsie is composed of the following episodes:—

I.—Premonitory symptoms. Auras.

II.—Stages of the attack proper.
- *a.* Epileptic period.
  - (1) Phase of tonic contraction.
  - (2) Phase of clonic contraction.
  - (3) Phase of resolution.
- *b.* Period of contortions and of "clownism."
  - (1) Phase of illogical attitudes.
  - (2) Phase of "grands mouvements."
- *c.* Period of passionate attitudes.
- *d.* Period of delirium.

III.—Terminal phenomena. Contractions, paralyses, &c.

M. Pitres remarks that all these phenomena are not seen in the attacks of ordinary hysteria. True as this description is in the cases from the study of which it was drawn up, there are marked differences between the attacks of common or "minor" hysteria, and of hystero-epilepsy or severer hysteria (la grande hystérie). The two affections belong to the same natural family of disease, but are not identical in the details of their manifestations. The convulsions of common hysteria are less complete and less regular. Certain phases of the hystero-epileptic attack are wanting. The epileptoid phase is very short, that of the passionate attitudes is absent, and the period of delirium, when present, ought to be placed rather amongst the closing

phenomena than in the active period of the attack. He thinks it useless to complicate the description of ordinary hysterical convulsions by seeking to identify them with those of hystero-epilepsy, and divides them into three stages only: (1) the pre-convulsive stage; (2) the stage of convulsions; (3) the post-convulsive stage.

I.—Pre-convulsive period.	1. Stage of psychical auras: the patients are anxious, restless, sad; they laugh or cry. 2. Stage of sensory auras: pains in various parts of the body, most intense in hysterogenic zones. 3. Stage of abdominal aura: a feeling of a ball, which rises from the ovarian, or sometimes from the epigastric region to the throat, causing a feeling of suffocation.
II.—Convulsive period.	1. Phase of tonic contraction: generally very short, the limbs becoming rigid for some seconds. 2. Phase of clonic convulsions: begins by one or several loud screams, followed by rapid, purposeless movements of different kinds; opisthotonus is frequent; intervals of arrest of convulsions, followed by fresh ones; the whole lasting 15 to 30 minutes or longer. From time to time the features are contorted into various expressions. Rarely the patients try to get up and escape from those who hold them.
III.—Post-convulsive period.	The patients remain for a certain time after the convulsions have ceased with eyes closed and limbs flaccid. After some minutes a talkative delirium begins. They may in this be either unconscious, or may reply momentarily to questions, lapsing again into delirium. This stage may be less obvious, the patient merely sobbing or laughing for a short time.

Finally, the delirium ceases, the patients appear for a moment astonished, and ask what has happened: after a short attack they resume their occupation, after a longer one they remain in a state of lassitude which makes them repose for some hours. They never pass into the state of stertorous sleep which succeeds an epileptic seizure.

The author divides hysterical seizures into (1) incomplete, in which one or other of the usual stages is absent; and (2) abnormal, in which the convulsive phenomena, attenuated or

modified in their symptoms, present very different characters from those commonly present in the regular attacks.

There are three varieties of "incomplete" convulsions; in the first, the pre-convulsive period, in the second, the post-convulsive, and in the third, both the pre- and the post-convulsive stages, are absent. The first variety is rare.

The abnormal attacks present the most varied symptoms, and sometimes closely resemble epileptic convulsions.

M. Pitres enumerates the distinctive features of the past history, symptoms of attacks, and general examination of the patients in the intervals, by which the differential diagnosis between epileptic and hysterical convulsions may be made. In doubtful cases, the examination of the urine passed in the twenty-four hours succeeding a fit, may give valuable information. After an epileptic fit, all the urinary constituents, particularly the urea, and phosphates are increased in quantity, and the proportion of the earthy to the alkaline phosphates, 1 to 3, remains normal; whilst after an hysterical convulsion the total amount of urea and phosphates is diminished, and the quantity of the earthy and alkaline phosphates excreted tends to be equal.

Contraction diathesis—"*diathèse de contracture.*"—Of this disturbance of the motor functions to which M. Charcot gave the name of diathesis, or state of liability to contraction, M. Richer says (97) "This state is neither paralysis nor contraction and yet partakes of the nature of both. Its presence is not betrayed by any objective sign: the patient preserves all his freedom of movement, and the manifestations of this condition require to be brought out either by the examination of the physician, or by some fortuitous circumstance. It is of the nature of paralysis in that it is generally concomitant with some muscular feebleness, and of the nature of contraction in that a very slight cause is sufficient to produce a contracture."

This state is common in hysteria; of 43 women and 27 men, M. Berbez (98) found 33 women and 19 men affected with it. It may be general or localized. M. Charcot observes that on the side on which a contraction develops there is almost always more or less anæsthesia, ovarian pain and some paresis, all of which have probably preceded the appearance of the contraction. Here we recognize an important law, that almost invariably disturbances of sensation of some kind exist in the domain of the contracted muscles, or are superimposed upon the contracture.

A simple means of provoking spasmodic muscular contraction is to tell the patient to squeeze the dynamometer with all his

force, the hand will then, in cases where the contraction diathesis is present, often remain contracted on the instrument.

The measures by which a contracture may be provoked in hysterical patients, are:—

(1) Excitation of the skin; light stroking, tickling, superficial electrisation, prolonged soft insufflation, &c.

(2) Excitation of the muscles: energetic voluntary contraction: pressure on muscular masses: repeated percussion of the muscles or their tendons; tetanisation by faradism; encircling the limb by a ligature: sudden flexion of a limb (paradoxical contraction).

(3) Irritation of bones or fibrous tissues; passive traction on joints, pressure or percussion on bones, &c.

(4) Psychical agencies corresponding to the different modes of suggestion in the waking state.

It is not necessary for the cutaneous excitations to be perceived for contracture to occur, they take effect on an anæsthetic limb. The contracture is not due to slight pressure exercised on the subjacent muscles, for it takes place when the excitation is made over the bone only, *e.g.*, the internal surface of the tibia. The spasm or contracture can be brought to an end by blowing upon the affected limb. When the spasm is generalised, or extends to a large number of muscular groups, it can be stopped by suggestion in hypnotizable subjects, or by provoking a convulsive seizure, either by means of pressure over the ovary, or by exciting the skin over the epigastrium. M. Pitres thinks that the fact that general muscular spasm can be induced by continued irritation of one spot shows that the stimulus acts through the ordinary reflex channels by the intermediary of the spinal gray matter. The persistence of the spasm, after removal of the stimulus, shows that it is not an ordinary reflex action, however, but rather is to be considered as the result of a lasting (durable) change of state produced in the gray matter by the action of the stimulus. Spasm may also be excited by applying (not too tightly) an Esmarch's bandage round a limb, *e.g.*, the forearm, for some time. After a little while, especially if the muscles are put into action by squeezing the dynamometer, spasmodic contracture of the muscles appears, putting the hand into the position of flexion from the predominating action of the flexors. If the bandage is applied too tightly the contraction ceases. When it is removed, as a rule the spasm disappears; if it persists, massage of the muscles will remove it. It may, however, become generalised. In this case, it may be limited to the muscles of the superior extremity only, or may spread to those of the

corresponding side of the face, trunk, and lower limbs: in this case there is generally anæsthesia of this side. Sometimes contractions may invade all four limbs and the whole face. When contractions have appeared spontaneously, or have been provoked on several occasions, they more readily become general. When a contraction, whether purposely excited or not, has lasted some time, massage often fails completely to remove it, whence the axiom of Charcot, "qu'il ne faut pas laisser traîner les contractures."

M. P. Richer has made the following observations on the electrical reactions of the muscles in the contraction-diathesis.

(1) In faradisation with rapid interruptions, the muscles stimulated enter into tetanus and other (neighbouring) muscles contract in addition to those directly excited. (2) If the interruptions are slow (3 per second) they give rise to contractions which, at first distinct from one another, are rapidly fused into a state of tonic contraction or tetanus. To single shocks (1) the line of descent (in graphic records) at first rapid, becomes slow, and then descends more rapidly to the base line. (2) Permanent contraction may be produced by a single stimulation; the line of descent is cut short, and the muscle remains shortened. (3) As a rule several shocks are necessary to develop a permanent contraction. After a few shocks the contraction begins to develop, then each new stimulation augments the contraction, which now does not remain limited to the muscle directly stimulated, but invades all the muscles of the limb. MM. Brissaud and Richet have shown (99) that a muscle already in a state of spontaneous or induced contraction will contract still further on electrical stimulation. It follows that in this state of contracture there is not the maximum shortening of the muscle, but an intermediate state between tetanus and relaxation; this myographic tracings demonstrate. M. Pitres has applied this fact in clinical work. "Remark," says he, "that the right arm of our patient has remained in the position in which it was placed before being invaded by the contracture. The fingers and the elbow which were semiflexed at the beginning of the experiment, remain so; the wrist which was in extension, is still extended. This proves that the contracture is not necessarily accompanied by any considerable or marked shortening of the muscular fibres."

This state of readiness to contraction is associated with muscular weakness. In some cases the weakness precedes the appearance of the contraction, in others it follows it. That

the muscular feebleness is not caused by the contraction-diathesis is proved by the fact, that the weakness is often seen to gradually improve, whilst the "diathèse de contracture" persists in power and in activity long after the limb has recovered its normal strength.

Together with the contraction-diathesis another phenomenon ought to be classed amongst the permanent stigmata of hysteria. This is a state of muscular feebleness or "amyosthenia;" it is often the forerunner of paralysis. It may be limited to a part of a limb, *e.g.*, the hand, may invade the whole limb or affect, which is common, a hemiplegic type. Sometimes all four limbs are affected. Whatever the seat or extent of amyosthenia it never involves one muscular group to the exclusion of its antagonists. No "active" deformities are thus produced by it. But in the case of the lower extremity, the foot may drop by its own weight, or that of the superincumbent bed-clothes, and from after a time become fixed in this position by adhesions in the joints and tendons. Care must, therefore, be taken to prevent this. As in the case of the contracture-diathesis, disturbances of general sensibility may also be present in the domain of the muscles invaded by paresis. "The tendon reflexes of an amyosthenic limb are generally exalted, and there often coexists this state of special aptitude to contraction" (Richer). M. Gilles de la Tourette, however, states that the paresis which accompanies the "diathesis of contracture" is independent of the "diathesis of amyosthenia" which is a clearly differentiated phenomenon, and that the reflexes in the latter are not, as a rule, exaggerated, but diminished. M. P. Richer says "that amyosthenia occurs most often on the left side; and that experiment has shown him that æsthesiogenic agents, which act on the sensibility, also affect amyosthenia, which is displaced, together with the anæsthesia which accompanies it, from one side of the body to the other, in the phenomenon of transference and its oscillations." This only applies when, as is the rule, sensory troubles are present.

Rhythmical hysterical spasms may be classified according to their seat as follows:—

(1) Rhythmic localized movements, limited to one muscle or group of muscles, and producing simple movements of flexion, extension or rotation of the head or limbs.

(2) Spasmodic co-ordinated movements of more complex character than the above, affecting several muscular groups synergically associated; hysterical chorea.

(3) Rhythmic respiratory spasms affecting the muscles of phonation and respiration.

The respiratory spasms may be divided into :—

(1) Simple, (*a*) inspiratory, (*b*) expiratory, the spasmodic movement occurring only during inspiration and expiration respectively.

(2) Mixed, in which both inspiration and expiration are modified by the spasm.

(3) Complicated, in which the muscles of respiration and phonation are both involved.

The rhythmical hysterical spasms must be distinguished from the "maladie des tics convulsifs" described by Charcot (100), Gilles de la Tourette (101), Guinon (102). This affection is hereditary, very obstinate, and charactised by the association of "tics" with diverse nervous affections such as coprolalia, the sudden involuntary utterance of filthy words; echolalia, automatic repetition of sounds or noises; echokinesis, repeated imitation of movements; fixed ideas; "délire du doute." Hysteria may sometimes simulate it but the diagnosis may always be made by studying the antecedent history of the patients, the conditions under which the illness developed, and the symptoms with which it is associated.

General characters of rhythmical hysterical spasms.

(1) Etiology. They occur generally in children or young adults, hereditarily predisposed to hysteria or already affected with it. They nearly always follow strong moral emotions, injuries, or an hysterical convulsion. The onset is sudden and the primary character maintained throughout.

(2) The spasms affect one or several muscles; sometimes groups of muscles habitually associated in their action, sometimes the muscles of one side of the body are involved. They may be quickly or slowly repeated, always at a regular interval. Generally sudden, like the muscular contraction following an electric shock, they may sometimes be slow and sustained. They may be constant or occur in series. They cease during sleep, and may be suspended by certain measures, compression of certain parts of the body, drawing off of the attention, reading aloud or singing, &c. They are not accompanied by mental disturbance, nor by loss of consciousness. The patients are perfectly aware of the movements but cannot stop them.

(3) The distinction of a rhythmical spasmodic affection of hysterical origin from one not hysterical is to be made by determining the presence of hysteria on other grounds; such as the

exciting cause of the spasm, history of former hysterical affections in the patient or in his family, and the presence of the sensory stigmata of the neurosis.

(4) The course and duration of these spasms is very variable. If the patient is in a situation where she is brought into contact with other persons who are predisposed to become hysterical, *e.g.*, in a prison or a convent, she should be isolated, or otherwise one case may lead to an epidemic outbreak amongst those who witness it.

In describing *hysterical mutism and stammering* Prof. Pitres gives the case of a young man who stammered from the time that he saw his father killed under his eyes, in an accident. This patient presented concentric contraction of the visual fields, and large areas of partial anæsthesia over the legs and the upper part of the trunk. M. Pitres thinks that many cases of ordinary stammering are really of hysterical nature, judging from the conditions under which the affection develops, namely, sudden fright, shock, or injury, and the suddenness with which cure often takes place.

Hysterical Paralysis.—The following characters distinguish hysterical paralysis.

(1) It is a flaccid paralysis with preservation of tendon-reflexes and of contraction of the muscles on mechanical and electrical stimulation.

(2) It is not transformed into spastic or atrophic paralysis. The muscles may waste after a time, but never to the extent observed in muscular atrophies of spinal origin, and the wasting present corresponds to the disuse of the muscles.

(3) It is accompanied by defects of common sensation and of the muscular sense.

(4) It often disappears during hypnotic sleep and reappears on waking.

(5) It can be relieved or displaced by agencies which have no effect on paralysis due to organic disease, *e.g.*, convulsive attacks, suggestion, application of "æsthesiogenic" agents.

Hysterical hemiplegia. Varieties:

(1) Partial hemiplegia, in which the arm and leg on one side of the body are affected, but the face escapes.

(2) Total hemiplegia, in which certain motor affections of the face co-exist with paralysis of the limbs on the same side.

(3) Hemiplegia with paraplegia, in which one arm and both legs are affected with or without participation of the face and tongue.

(4) Alternate hemiplegia, paralysis of the limbs on one side, and of the opposite side of the face.

(5) Double hemiplegia, in which all four limbs are paralysed.

(i.) The first variety has been well described by Todd "The face and the tongue escape; the paralysis, limited to the arm and leg, is often incomplete; the muscles are always flaccid and are not wasted; sometimes one or other of the paralysed muscles is the seat of muscular spasms or of cataleptic rigidity. In walking, if the paralysis is well-marked, the leg is dragged like a foreign body, and scrapes the ground."

(ii.) The total hemiplegias show a remarkable peculiarity, the face and the tongue, instead of being paralysed are the seat of spasmodic contraction, known from the researches of Charcot as hysterical glosso-labial hemispasm. This symptom is, according to Charcot, characteristic of hysteria.

Hysterical glosso-labial hemispasm.—In repose the mouth is asymmetrical, the labial commissure is drawn upwards and outwards, and the naso-labial furrow deepened on the affected side. The tongue lies on the floor of the mouth, deviating towards the affected side, and often cannot be protruded. When the patients can protrude it, it is strongly deviated and curved ("en crochet"), its tip directed away from the side of the facial spasm and paralysed limbs. M. G. Lombroso (103) has reported some cases of hysterical hemiplegia with paralysis of the lower part of the face, as in organic hemiplegia, but these require confirmation, and at present this glosso-labial hemispasm must be regarded as one of the most important symptoms of hysterical hemiplegia.

MM. Brissaud and Marie describe in the following terms, after the teaching of M. Charcot, the differences between hemiplegia of hysterical and organic origin. "In patients suffering from a localised organic cerebral lesion, the paralysed leg, instead of being, as in the normal state, carried directly forwards by flexion of the leg and thigh, can only be brought in front of the other by a movement of abduction, in which it describes a semicircle of which the sound foot is the centre. During this movement, the paralysed limb remains extended. In hysteria, on the contrary the paralysed leg is no longer brought forwards, but is dragged behind the sound limb. The patient walks in something the same way as children go upstairs, always with the same leg first. Further, during walking, the paralysed limb is often a little flexed, and not extended as in the case of organic hemiplegia."

(iii.) In alternate hysterical hemiplegia the side of the face or tongue opposite to the paralysed arm and leg is affected with spasm (glosso-labial hemispasm).

The most common complications of hysterical hemiplegia are œdema, muscular atrophy, and persistent (permanent) contractions of the paralysed limbs.

Hysterical Paraplegia. Varieties.—(1) True paraplegia, complete loss of power in the legs in all positions; (2) "abasie," in which the paralysis only appears when the patients are in the vertical position. (1) Hysterical paraplegia may be of the flaccid or spastic kind.

(2) Under the name of "abasie" is described a form of hysterical paraplegia, in which the patients when lying down are able to perform any movement with their legs, but when standing are powerless.

MM. Charcot and Richer (104) described, in 1883, this special form of pseudo-paralysis of the legs, and M. Blocq gave a more extended account of it, and showed that this affection is ordinarily closely connected with hysteria, in the course of which it forms an episode, generally a passing one (105). It most frequently occurs in boys of 8 to 14 years of age, but may be seen in adults and in the aged. It comes on either slowly or suddenly; supervening generally on a moral emotion or slight injury. It is characterised essentially by the loss of the co-ordinated muscular movements by means of which equilibrium is maintained during standing and walking. When the patients sit or lie they can move their legs normally, there is then no loss of muscular power nor of the muscle-sense; the knee-jerk is normal, there is no ankle-clonus. When they try to stand or walk, their legs bend under them; they are incapable of holding themselves upright, and of moving forwards (abasie paralytique), or in other cases, their legs are the seat of tremors (abasie trepidante), or execute inco-ordinated movements resembling those of ataxics (abasie choréiforme). There are several varieties. M. Charcot classifies the cases as above (106). The divisions might be multiplied, but this would needlessly complicate the study of the disease. The acts of walking, leaping, climbing, dancing, &c., are all complex acts which we have acquired by long training. Each of them implies the co-ordinated action of groups of muscles associated by education to act together for this end. Each of these actions can be performed independently of the others, and corresponds to a distinct muscular mechanism which can be separately deranged. Just as a man can walk without being able

to dance or swim, so a patient may lose the power of walking, and yet be able to jump with his feet together, or to go on his hands and knees. Very clear examples of this are afforded by different cases of abasie (107). The prognosis is generally good. "Abasie" has lasted in reported cases from some days to fifteen months. Recovery is the rule, but relapses are common. M. Blocq thinks that the functional disturbance which produces "abasie" may be either an inhibition of the controlling cortical centre which puts into action the spinal mechanism that presides over the act of walking, or an inhibition of this spinal centre itself.

Hysterical pseudo-tabes.—In pseudo-tabes the characteristic lesion of the posterior columns of the cord is absent, but the patients present more or less faithfully the classical motor and sensory symptoms of locomotor ataxy. These symptoms may be due to cerebellar tumour, to the lesions of disseminated sclerosis, to peripheral neuritis, or lastly may not depend upon any organic lesion of the spinal cord or nerves as in certain cases of hysteria and of neurasthenia.

Hysterical and neurasthenic pseudo-tabes present the same group of symptoms.

There are on record ten well authenticated cases of hysterical pseudo-tabes, of which six were men and four women. The onset is slow, the course progressive, and the duration varies from some days to several years. In some cases temporary cure was followed by a relapse.

Symptoms.—The most constant were, instability in standing and motor inco-ordination in walking, increased on closing the eyes. The gait often exactly resembled that of true tabes dorsalis. Lightning pains occurred in half the cases; girdle pains were almost as frequent. There were also spinal pains and tenderness to pressure over the vertebral spines. Paræsthesie were sometimes noted in the lower extremities. In some patients there was hyperæsthesia, in others anæsthesia of the soles. The sense of position of the limbs was preserved. The plantar reflexes and knee-jerks were present. There was no Argyll-Robertson phenomenon nor atrophy of the optic discs. In one or two patients there were slight bladder and rectal troubles. The patients often presented in addition to the above symptoms the sensory stigmata of hysteria or the characteristic symptoms of neurasthenia. Hysterical pseudo-tabes then resembles true tabes dorsalis in the disturbance of equilibration and of walking, in the presence of Romberg's sign and of troubles of sensation, but differs from it in

the preservation of the knee-jerks and plantar reflexes, and in the absence of optic atrophy and of the Argyll-Robertson phenomenon.

Hysterical Tremors.

Contrary to the opinion of Briquet, who considered that hysterical tremors were a "convulsion en petit," M. Gilles de la Tourette thinks that they form part of the common basis of hysteria formed by the union of the permanent stigmata. Like the sensory troubles these tremors show great tenacity, persisting during several months, and at times intensifying so much as to form "attacks of tremor." These paroxysms are only the exaltation of the stigmata, and if the contraction-diathesis and the sensory troubles are accepted as permanent stigmata, tremor must be equally so accepted.

Many cases of hysterical tremor had been published but remained isolated until M. Charcot (108) undertook a systematic account of the subject, and was followed by Prof. Pitres (109), M. Rendu, and lastly by M. Dutil (110), in whose inaugural thesis the fullest account of the subject is to be found.

Their frequency is less than that of the disturbances of sensation or the contraction-diathesis, but further observations on this point are needed. M. Charcot teaches that hysterical tremor especially occurs in men, and is often present in cases of traumatic hysteria; meanwhile it is frequently seen in women and is not rare in children. Tremor, whatever form it may take, generally appears suddenly under the influence of a traumatism, fright or moral shock of some kind, and very frequently after a convulsive attack. When it exists previously, the occurrence of an attack increases it. It may be general or partial in distribution, may affect a whole limb or part of it, or assume a paraplegic or hemiplegic form. The duration is very variable. Tremor may last for months or years without any interruption. It may be slight or so severe as to render walking difficult and the ordinary uses of the arm and hands almost impossible. Classical authors say that paralysis agitans and senile tremor sometimes supervene suddenly on a strong moral emotion: the question of hysteria in such cases requires fresh investigation in the light of recent observations.

The tremor has generally a regular rhythm but the frequency of the oscillations is very variable, even in the same patient at different times. It may be remarkably slow, very rapid, or of medium rapidity, this latter being the most common form.

The following are the classifications adopted by M. Charcot and by M. Dutil; they do not differ essentially.

M. Charcot's classification.

A. Tremor, exaggerated by voluntary movements.	1. Oscillating tremor with oscillations 3-6 per second.	Resembles paralysis agitans or senile tremor.
	2. Oscillating tremor with rapid oscillations 8-9 or more per second.	Resembles the tremor of Graves' disease, of alcoholism and of paralysis agitans.
B. A tremor, which may or may not exist during rest, is provoked or exaggerated by volitional movements which do not accelerate it, but increase the amplitude of the vibrations.	3. Intentional tremor (type Rendu) intermediate between 1 and 2 with regard to the frequency of the oscillations.	Resembles the tremor of disseminated sclerosis and still more perfectly mercurial tremor which according to M. Letulle is very often of hysterical nature.

M. Dutil's classification.

1. Vibratory tremor 8-9 oscillations or more per second.	Persists during rest, is little or not at all modified by voluntary movement.	Resembles tremor of Graves' disease, of alcoholism, and of general paralysis.
2. Tremor of medium rhythm 5½-7½ oscillations per second.	*a*. Intentional remitting tremor (type Rendu). It may or may not exist during rest, and is exaggerated during voluntary movement.	Resembles exactly mercurial tremor and less perfectly that of disseminated sclerosis.
	b. A paraplegic form of the above.	Resembles the "spinal epilepsy" of spastic paraplegia.
	c. Purely voluntary tremor, only appearing on voluntary movement.	Exactly resembles the tremor of disseminated sclerosis.
3. Slow tremor 4-5½ oscillations per second.	Persists during rest, is little or not at all modified by voluntary movements.	Resembles paralysis agitans and senile tremor.

The vibratory tremor No. 1 may be generalised or partial in distribution and as a rule lasts only for an hour or two being then only a post-convulsive phenomenon. In some patients, however, it may become chronic, and when well marked and general the patient is agitated by a constant trembling or vibration whether he is standing or sitting. When stripped the muscles of the trunk and limbs are seen to be agitated by little fibrillary

contractions, which in the fore-arms may determine small sudden movements of the fingers. When the tremor is at its maximum intensity as after a fit or some strong emotion the muscles of the face and lips share in the tremor, the speech is tremulous, and the aspect of the patient exactly recalls that of a person affected with general paralysis. In the quieter periods the tremor resembles that of Graves' disease. Periods of quiet alternate with periods of increase of tremor, which only entirely ceases during sleep. In the common run of cases, this tremor does not interfere with ordinary movements, except those which require a certain amount of precision such as writing, so that walking is ordinarily not interfered with, but during the exaggeration of the tremor which follows a convulsive attack a giving way of the legs occurs, and being frequently repeated, renders standing very difficult. Graphic tracings show that the oscillations are small, restricted in range, and of unequal amplitude.

The first variety of the tremors of middle range—intentional remitting tremor—resembles that of disseminated sclerosis but differs from the latter in being constant, meanwhile it may cease in the sitting or lying posture and during sleep. Is exaggerated during volitional movements. The patient's head, trunk and limbs are agitated by constantly repeated small rhythmical muscular contractions. If the patient is seated the head nods to and fro, the forearms and hands are agitated by slight movements of flexion with pronation followed by extension; whilst the feet tap against the floor with a noise resembling that made by an impatient person who taps with his toes. A voluntary movement of the limb exaggerates the tremor; if a glass of water is carried to the lips the water is spilt by the progressively increasing tremor; writing and similar fine exact movements are often rendered impossible. When the patient stands the oscillations become greater and more abrupt, and constant slight movements of flexion and extension of the legs at the knee occur. This tremor generally predominates in the limbs on one side of the body. It is increased by convulsive attacks, emotions, and irritation of hysterogenic zones. It may persist for years, or pass away more or less rapidly. Though further observations are necessary to form a conclusion, there is much evidence that mercurial tremor, which this variety closely resembles, is in the majority of cases of hysterical origin.

The second variety of the tremors of medium amplitude only differs from the foregoing in being confined to the lower extremities.

The third variety "purely voluntary tremor" exactly re-

sembles that of disseminated sclerosis. How is the diagnosis to be made between the latter disease and the hysterical manifestations which simulate it, and are accompanied by this peculiar tremor? The difficulty of diagnosis is often very great; no general rules can be given, each particular case must be considered on its merits. The presence of stigmata and a past history of hysteria, will be in favour of the neurosis, but it must be borne in mind that hysteria and disseminated sclerosis may be associated in the same subject, for M. Charcot has shown that there is no organic cerebro-spinal affection with which hysteria is more frequently combined than this one. Several cases have been published by Westphal, Langer, Babinski, and others, in which the diagnosis of disseminated sclerosis was made during life, but no lesion of any part of the nervous system was found at the autopsy. M. Gilles de la Tourette thinks that such cases should be placed under the category of hysteria. He further remarks "that this view has been recently combated by Dr. Buzzard. . . This author . . . recalls that a number of organic affections have been taken for hysteria . . . and insists on the diagnostic value of the *intentional* tremor which, he says, does not exist in hysteria. After the facts we have brought forward, we shall be permitted to say that this opinion is no longer tenable. And Dr. Buzzard concludes by saying that many symptoms attributed hitherto to hysteria belong to organic affections, and that the domain of the neurosis is destined to be limited, as the measure and extent of our knowledge of organic affections of the spinal cord increases" (111). On the other hand, M. Souques working at the subject from the opposite point of view (112), says: "We sustain here exactly the reverse thesis, and we allege that, in proportion as we become better acquainted with hysteria, so a number of unusual cases, referred to the organic diseases, will be found to belong to it. As for the cases where during life symptoms of hysteria have been noted, and referred to the neurosis, and where the autopsy has revealed organic lesions, we have obviously to do with those associations of hysteria with organic disease on the frequency of which M. Charcot has for several years insisted."

The third variety of tremor (of M. Dutil) is made up of slow oscillations, and almost exactly resembles the tremor of paralysis agitans. The general aspect and bearing of the patient may also strikingly simulate that of patients affected with the latter disease. These cases are, however, not very common.

All forms of hysterical tremor cannot be brought under the varieties of the foregoing classifications; some being variable

and changeful in character. They may be accompanied by choreic movements, and so simulate the tremor and choreiform movements which occur in certain cases of cerebral tumour. Lastly, there may occur in "normal hysteria" muscular contractions which may affect, separately or together, the trunk, head, or limbs. But these "rhythmical spasms" occur so often under the form of attacks (rhythmical chorea) that it will be better to consider them with the convulsive paroxysms of hysteria.

Trophic disturbances in Hysteria.—Œdema generally appears as a complication in contracted or paralysed limbs, but may rarely come on independently of any affection of motion. Sydenham pointed out the characters of the œdema of hysteria as being more marked in the morning than in the evening, as not pitting on pressure with the finger tip, and as often affecting only one limb. M. Pitres thinks that this exaggeration of œdema in the morning is not constant, and cannot be relied upon as a diagnostic distinction between hysterical œdema and that of cardiac or renal origin.

As a rule, hysterical paralyses are not accompanied by any alterations in the electrical reactions of the muscles. Sometimes, however, the paralysed muscles rapidly lose their excitability to both faradic and galvanic currents. It is even possible that, as in a case reported by MM. Gilles de la Tourette and Dutil, the reaction of degeneration may be present. Prof. Pitres has not seen an instance of this, but relates a case of hystero-traumatic paralysis of the right side of the face following a burn. The affected muscles showed marked diminution of excitability to both forms of current, but no reaction of degeneration; this was noted fifteen days after the onset of the paralysis, and did not stand in the way of a speedy cure. There was also spasmodic contraction of the elevators of the lower jaw, which almost completely prevented opening of the mouth. It has already been observed that in hysterical hemiplegia the facial muscles behave differently from the other muscles of the body, and it is curious that in this case of hystero-traumatic paralysis of the face some muscles were contracted, whilst others were paralysed. This association of paralysis and contracture may prove useful in diagnosis. Local destruction of tissues from trophic lesions are rare in hysteria; but the nails may spontaneously fall off, and the author relates a case which he thinks shows that the teeth may be lost, and a sacral bedsore form.

Nutrition in Hysteria.—Even till lately it was generally accepted as true, that although hysterical patients eat a very in-

sufficient quantity of food, and often suffer from digestive disturbances, their general state of nutrition remains good. M. Empereur (113) arrived at the conclusion that, although the quantity of food taken was insufficient, wasting did not occur because disassimilation was sluggish; the outgoings, the loss due to disassimilative tissue change, were small, and, therefore, in spite of the small quantity of food taken, the general nutrition was maintained.

MM. Gilles de la Tourettte and Cathelineau have made observations on the state of nutrition in "normal" hysteria. They define the "normal" hysteric as a patient, who presents at the time of observation, no other manifestations of the neurosis than the permanent stigmata which mark him as the subject of confirmed hysteria. They selected, to put themselves above criticism, patients who had entered the hospital for hysterical convulsions, contractions, cough, mutism, &c., and were careful to make their experiments at a time when these symptoms could not influence the nutrition, *i.e.*, during the interval between widely separated attacks. The conditions under which the patients at the Salpêtrière live are, from the vast extent and general arrangements of the hospital, those of ordinary life. They found first that "normal" hysterical patients took a quantity of food quite sufficient to nourish perfectly a healthy person living under the same conditions. In their observations, 10 patients, 7 women and 3 men, were taken, their weight was ascertained, and the temperature taken every two days. The urine was collected from each patient for the twenty-four hours, measured, and at once analysed. The patients were directed to pass water before going to stool. During the period of observation, eight days in each case, the patient was placed under close surveillance. They found that in the hysteric, apart from manifestations of the neurosis other than the permanent stigmata, the nutritive processes are normally carried on, and the volume of the urine, and the urinary excretra bore the same proportion to the body weight as in the healthy.

From researches on the blood he concludes that—

(1) In a "normal" hysteric, the same solution of continuity of the integument as that made in a healthy person, gives issue to a quantity of blood about one-third less than the normal.

(2) Putting aside cases of anæmia and chlorosis, the number of the corpuscles, the quantity of hæmoglobin, urea, and glucose in the blood are normal.

The mental state of hysterical patients.—It is necessary, says

M. Charcot, to take hysteria as it exists, that is to say as a disease par excellence psychical. The ordinary mental state of hysterical patients must be considered, their manner of being, their mode of action, their reaction to their surroundings, their whole moral and intellectual behaviour. Most authors even up to quite recent times represent the hysterical woman as of an extraordinarily complex type, exceedingly versatile, remarkable for her spirit of duplicity, lying, and dissimulation. One common trait characterises them, said Tardieu, namely, instinctive simulation, an inveterate and constant necessity of lying without interest and without object, for the mere sake of lying. According to these writers, the psychologic state of hysteria includes almost all mental pathology, from the most evident acute delirium to the most characteristic chronic monomania. The labours of Charcot and of his pupils have done as much for the psychical as they have for the physical phenomena of hysteria. Nervous heredity is strong in the hysterical patient and if it is often homologous —mother hysterical, daughter hysterical—there is often also found in the family history evidence of insanity, epilepsy, &c. The conclusion follows that hysteria may, perhaps, more than any other disease, beyond its own proper mental state present the psychical stigmata of "degeneration" (dégénérescence).

When the errors of observation and false statements that have obscured the actual mental state in hysteria have been cleared away we find that the true condition becomes clear and may be expressed in one word, suggestibility, that is, extreme sensibility to suggestion. But if this condition of suggestibility is the one mental state it is not the less a complex one. The suggestions may come from outside, be extrinsic, or be intrinsic, the latter constituting auto-suggestion, a factor of extreme importance in hysteria.

What is the mechanism of this suggestibility? The convulsive attacks play a large part in it. The prodromal period must be included in these attacks; it may last for 1-3 days or longer, and throughout its duration the patients are restless and agitated. Some are excited and contentious, or laugh and cry without any reason, others are depressed, and seek solitude. Then come the convulsions, on the other hand the convulsion may abort, especially in children in whom, as M. Charcot has shown, it may take the form of mania and be composed of psychical prodromata with some contractions of the arms or twitchings of the eyeballs to alone represent the convulsive period.

These prodromata are not accurately speaking part of the

mental state of hysterics, they belong to the paroxysm, but in certain patients a succession of attacks may create a sort of permanent mental state. When these psychical manifestations appear, one tries to induce the threatening attack, and if the convulsive phenomena are not produced and the mental troubles persist for more than the usual time one can say that the attack will not come; it has had its psychical equivalent. In children this often occurs, but in the majority of cases the prodromata are accompanied by the delirium which characterises the fourth stage of the convulsive paroxysm. This delirium singularly influences the mental state in the intervals of the attacks. A profound impression—on account of the memories revived, for the delirium is most often a recalling of past acts of the life—is exercised on the brain by this delirium, and the hysterical patient is possessed for several days after the attack by this impression, which is generally a painful one. The hallucinations which end the attack are frequently so vivid as to give the patient the illusion that imaginary events have actually occurred. We have seen one of our patients, a man, after the end of the attack, get up suddenly, open the window and chase the purely visionary black cat that had tormented him. This delirium may directly produce physical troubles, as for instance, paraplegia in a patient who has believed himself to have fallen down a precipice. Numerous examples of this kind may be found in the history of epidemics of demoniac possession. On the other hand many phases of the delirium, which can be recognized by the attitudes and gestures of the patient at the time, are not remembered when she wakes; they have however modified the mental condition and the patient is in a state of joy or sadness, for which she can give no reason. Similar central processes explain the "*lacunæ of memory*" found in hysteria, through which a whole period of the past life may be forgotten one day to be recalled the next. Hallucinations of vision are particularly common, and have one characteristic which M. Charcot has brought out. The phantoms are not motionless but move in a direction which is always the same. The cats, rats, &c., run along passing before the patient from left to right, or from right to left, according as the hemianæsthesia affects the left or the right side. The hallucination always takes its departure from the anæsthetic side. Most often the phantom passes by the side of the patient; it comes from behind her to vanish in front of her on the *anæsthetic side* (114).

If auto-suggestion was only effected under the influence of attacks, the phenomena that it brings with it, and which occupy

so large a place in the mental state of hysteria, would be easy of interpretation. But we must take into consideration those cases of hysteria in which convulsive attacks are either absent or occur at long intervals. In this connection, the terrifying dreams and nightmares by which the sleep is disturbed in hysteria, and which Sydenham described, are of great importance in understanding aright the mental state. These nocturnal hallucinations are analagous to those of the attacks, but are much more frequent, occurring every night, whilst attacks are relatively rare. Further, the attacks can hardly pass unnoticed, whilst the hysteric is alone the witness of her dreams, and her friends do not know to what to attribute her changeful character, influenced as it is by the nature of the dreams that are often forgotten by morning. The instability of the hysterical temperament may be explained in this way, by its varying from day to day, according to the nature of the nocturnal hallucinations and their influence on the conduct of the following day. Paraplegia even may be determined by a prolonged dream, of which Féré has reported a remarkable instance (115). M. Pitres has also shown that the nocturnal hallucinations, like those of the attacks, may be accompanied by persistent painful sensations. "One of our patients receives, from time to time, an imaginary visit from an old woman, who touches her in different parts of the body, and on all the parts touched, hyperalgesic zones develop which last for several days."

The intensity of these dreams may even determine trophic troubles; and the author relates the case of a girl of 19, who, after being disturbed by terrifying dreams during the night, felt towards morning a sharp pain on the inner side of the right leg, and when dressing found there an oval patch of "redness:" there was also right hemianæsthesia especially marked in this leg. He attributes this subcutaneous hæmorrhage, in the absence of any evidence of a traumatism, or of a nocturnal fit, or of its existence on the previous evening—to the influence of a very vivid dream. A passage from the life of St. Theresa is quoted to show that she was similarly the subject of painful hallucinations.

The nocturnal dreams and hallucinations in the female, are sometimes of an erotic character; this is much less often the case in men, though they may be as much tormented by dreams as women. To the persistence of these hallucinations in the waking state is to be attributed all these stories of "incubi" and "succubi" which are so prominent in the epidemics of demoniacal possession in the middle ages. These erotic hallucinations are

generally accompanied by severe pains. From the medico-legal point of view, the importance of these erotic hallucinations is obvious, as innocent persons have been unjustly accused and condemned on evidence of this kind.

Auto-suggestions, similar to the preceding, which are derived from the hallucinations of attacks or of dreams, may arise during the waking state. In this case, they are associated very often with nocturnal hallucinations. The extreme impressionability or suggestibility of hysterical patients has been noticed by most authors, and some very striking examples of it are given by M. Charcot (116). The hysterical also show an extraordinary credulity, and in a general way resemble children. The troubles of sensation, the stigmata, have an influence on the general bearing of hysterics. If, for instance, a patient likes to eat raw onions and lemons, it may be because ordinary food does not affect her anæsthetic palate; nor does the conduct of the man, who walked in winter in the courts of the hospital, clad only in a shirt and waistcoat, seem so eccentric when it is remembered that he suffered from total anæsthesia, and that cold did not affect him.

With regard to the question of simulation in hysteria. The true malingerer is a scheming, active person; the hysteric who malingers is not conscious of simulation. She is a passive being, a photographic-plate which has registered impressions, and keeps them as she has received them, sometimes indeed amplified, but always unconsciously and in good faith. Certainly malingerers exist, and may be hysterical, but they do not simulate from the fact of their being hysterical. The hysterical brain does not lend itself to enduring combinations; it is the slave of the suggestion of the moment, the mould on which an unconscious suggestion forms, and if the hysteric sins against the laws of society, it is very often beyond her own proper act. M. Laurent has well said (117): "How does the hysteric become a criminal? Rarely of his own will. The initiation of the crime by no means generally comes from him, almost always a more powerful will has prevailed over his own. He is *par excellence* the being who responds most readily to impressions from without, and obeys them without restraint." Noteworthy is the indifference that some hysterical patients, who are suffering from a local manifestation of the neurosis, paraplegia for example, show to it. Once affected, they seem not to concern themselves about it any longer, and under these conditions it does not fail to persist. In such cases, in order to effect a cure, it is necessary that "one suggestion should oust the other." M. Charcot has

shown the nature of this process of cure, and of the mode of action of isolation (118); he has also pointed out that, even in the most marvellous cures the curative suggestion operates generally only incompletely, and not immediately. A paralysed arm, for instance, does not suddenly regain its normal muscular power, even when trophic disturbances are absent, and the sensory stigmata persist a longer time still after the motor troubles have disappeared.

Hysteria may be associated with mental degeneration, but hysterical insanity has no real existence. Beyond the peculiar mental state that we have described, with its sudden suggestions due to extrinsic suggestions or to auto-suggestions, under various forms of hallucination, there exist only states of delirium or prolonged "somnambulistic" periods, which in no way resemble chronic insanity. So we may, *a priori*, conclude that the hysterical patients in asylums have been placed there for acts arising from their mental degeneration, and not from the hysteria associated with it. Numerous examples sustain this view (119).

In children, hysteria takes, in the majority of cases, a transitory character; the patients are extremely impressionable, the affective element predominates. The brain is, in children, in the period of rapid evolution; impressions received are strongly felt, but do not leave, as a rule, persistent traces.

In male hysteria, the mental state in those who have become affected by the neurosis in infancy or adolescence, is one in which a morbid "suggestibility" is dominant. The general bearing may be said to be effeminate. They are romantic; often boasting themselves of sexual powers which are imaginary.

Another type is more common in these cases, in which the psychical state of neurasthenia is added to that of hysteria. Intellectual impairment, cerebral apathy co-exists with lapses of memory, which render all work impossible. These lapses of memory are remarkable enough to lead to a suspicion of lying or malingering.

This mental state of hystero-neurasthenia is not peculiar to men, but is found in women, in whom the manifestations of hysteria first appear at the menopause. In old age, the hallucinations, which formerly existed and bore witness to an excessive cerebral activity disappear, and the mental condition is one of sadness and melancholy.

On suicidal attempts in Hysteria.—Many authors have considered the suicidal attempts made by hysterical patients as not serious; they think that the latter have no intention of actually

carrying them out, but make their preparations with the view of attracting sympathy from, or evoking astonishment in, their neighbours (120). They hold that, generally speaking, the suicide of hysterics is a comedy, an act of simulation skilfully considered and prepared. M. Pitres, however, analysing their own observations shows that even these are sometimes in contradiction with this theory of simulation, as several of the attempts at suicide terminated in death. He then gives an account of eleven attempts at suicide in his 100 patients, and concludes that in hysteria suicide is generally the result of a sudden determination, taken without reflection, but that there is no sufficient reason to consider it as a comedy, played by the patients to make themselves interesting and to alarm their friends and neighbours. If the attempts at suicide made by hysterics do not often end fatally, the reason is that they are commonly not premeditated. The melancholics who desire to die, select a long time in advance, the moment and the means favourable for the execution of their project. The hysteric does not reflect. For a mere contrariety or trifling disappointment, she takes the resolution of killing herself, and thereupon swallows any poison that is at hand, or throws herself into the water. Next day, she is delighted at having escaped death. Facts of this nature are to be explained by the puerility of the hysterical character, passing as hysterical patients do like children, in one moment from grave to gay, from gentleness to violence, or from love to hate. With regard to the question of simulation, Prof. Pitres thinks that it is much less frequent than has been and is now stated. Hysterical patients relate what they feel without regard to the consequences. The unbridled love of deception which has been attributed to hysteria is a legend without serious foundation, as common sense and clinical observations show. It is impossible to suppose that the so-called witches who went to the stake for having stated that they had ridden to a "Sabbath" on a broomstick, or that, as has happened in our days, a patient, who had an hysterical club-foot amputated, could have been lying, in the first case, merely to gain notoriety, in the second, to enjoy the credulity of the surgeon.

General treatment of Hysteria.—Prophylactic treatment is very important; when a child shows the first signs of a neuropathic constitution, the symptoms of which in childhood have been already mentioned, the morbid tendency should be corrected by judicious physical and moral education. All forms of exercise which develop the muscles and strengthen the body are useful. Intellectual over-work, violent emotions, *e.g.*, terrifying stories of

ghosts, or giants, in short, everything which excites their sensibility and imagination is harmful. Children predisposed to hysteria are far better brought up away from their parents at a good school.

Before undertaking any plan of treatment the physician should fully gain the confidence of his patient, he should assure him that the malady from which he is suffering is not a grave one. That his symptoms are not uncommon, that they will pass away after a short time even without treatment, but that they will disappear more quickly on the carrying out of proper therapeutic measures. When the patient is surrounded by affectionate and at the same time injudicious friends, who are terrified at the least incidents of the malady and confirm him in his belief that he is suffering from an extraordinary and incurable disease (parents are often the most injudicious in this respect), then *isolation* from the friends is of the first importance. When the patient is isolated he will be placed in the most favourable position for deriving advantage from further treatment which should be directed (1) to bring to an end as speedily as possible the actual symptoms of the neurosis present and (2) to combat the morbid susceptibility from which these symptoms arise.

The measures to be employed for the second object are:—

Hydrotherapy, which is one of the most powerful agents in nervous affections. It may preferably be employed in the form of a cold douche, over all the body for a duration of 20-40 seconds, followed by a jet of hot water over the feet, and energetic rubbing with a dry towel. If this is not borne warm baths at 34°C for 45-60 minutes, or colder baths (30-28-26°C) of 5-15 minutes duration may be employed.

Static electricity, recommended especially by Prof. Charcot, demands costly apparatus and has other disadvantages. Drugs are very rarely of any use. Iron, arsenic and quinine are useful when chlorosis is present but have no direct effect on the neurosis. Morphia is undoubtedly effectual but unless used with great caution the remedy may be worse than the disease.

As for hypnosis (121) paralyses, contractures, rhythmical spasms sometimes disappear with marvellous rapidity under the influence of suggestion, but it does not cure the hysteria itself. All hysterical patients cannot be hypnotised, and of those who can be hypnotised all are not amenable to suggestion. Further this method is not always harmless; certain subjects become, after some séances of hypnosis, so sensible to hypnotisation that a

look, gesture, or unexpected noise is sufficient to throw them into a state of hypnotic sleep. In short, hypnosis is a mode of treatment difficult to manage and which should be reserved for special cases. If a patient has been previously hypnotised by others or is subject to attacks of (hypnotic) sleep, hypnotic suggestion may be tried. The author does not think it well to go further, nor to commence treatment by hypnosis unless at the most pressing request of the patients or of their friends.

None of the operations on the generative organs which have been proposed and carried out are justifiable.

If asked as to the question of marriage as a cure for hysteria it is well not to take the responsibility of advising it.

Additional Note on Nutrition in Hysteria.

We may add here that the statements of MM. Gilles de la Tourette and Cathelineau, which have been quoted above, as to the changes in the urinary excreta after paroxysmal manifestations, have been criticised by other observers, and the question of the value of these changes in differential diagnosis must, perhaps, at present be regarded as *sub judice*. M. Voulgres, in a thesis (122) on this subject, finds that intellectual work and fatigue increase the secretion of the earthy, muscular work that of the alkaline phosphates, so that in a healthy man fatigued by a series of efforts, there is a tendency to inversion of the normal ratio. He agrees with the above authors that the proportion of the earthy to the alkaline phosphates tends to become, 1-2 equal, or even 2-1 during the twenty-four hours following an hysterical convulsive seizure, but says also that there is a similar, but less marked tendency, in non-convulsive hysteria, the proportion being 3 to 1.5. He further states that this inversion of the normal formula is not a pathognomonic sign of either convulsive or non-convulsive hysteria, for he has found it in other diseases, in paraplegia epilepsy, neurasthenia, locomotor ataxy, and phosphatic diabetes without nervous symptoms. He concludes that inversion of the ratio of the earthy to the alkaline phosphates is the *rule* after an hysterical, the *exception* after an epileptic fit.

NOTES AND REFERENCES.

(1) *Celsi de re medicâ.* Lib. vii., cap. i., sec. vii., p. 213. Paris: Didot, 1872.

(2) *Traité des maladies nerveuses ou vapeurs et particulièrement de l'hystérie et de l'hypochondrie.*

(3) Who considered hysteria as a general malady of nervous origin affecting both sexes.

(4) Who denied its existence in men and upheld the theory of its origin in the uterus.

(5) *Lectures illustrative of certain local nervous affections*, London 1837.

(6) *Op. cit.*, p. 82.

(7) Hammond. *Treatise on the Maladies of the Nervous System.*

(8) *A System of Medicine*, edited by Russell Reynolds, *art. Hysteria*, vol. iii., p. 306.

(9) *Contributions à l'étude de l'hystérie chez l'homme*, p. 25. Paris, 1885.

(10) *Hysterie beim mannlichen Geschlecht, Inaug. Diss Wurtzbourg*, 1889.

(11) *Thèse de Bordeaux*, 1890, p. 135.

(12) *Op. cit.* p. 134.

(13) *L'hystérie, à la consultation du Bureau Central des Hôpiteux de Paris, Etude Statistique. Progrés Médical*, 27 July, 1889, p. 28).

(14) *De l'hystérie mâle dans un service hospitalier. Arch. Gén de Médecine*, Aug. 1890, p. 168.

(15) Moran. *Compte rendu du service ophthalmologique pour l'année* 1888. *Archiv. de Neurologie*, 1890, p. 436.

(16) *Leçons sur les maladies du système nerveux*, vol. iii., p. 226, 1887.

(17) *On railway and other injuries of the nervous system.* London, 1866.

(18) *Hysterical anæsthesia brought on by a fall. Boston Medical and Surgical Journal*, 11th December, 1884.

(19) *Archiv. für Psych.*, vol. xv.

(20) *Leçons sur les maladies du système nerveux*, vol. iii., 18th, 26th chapters.

(21) Charcot. *Mal. du Syst. Nerv.*, vol. iii.

(22) *Gaz. des Hôpitaux*, 1885.

(23) *Contribution a l'étude de l'hystérie chez l'homme.* Th. de Paris, 1877.

(24) *Hémianesthésie saturnine et hémianesthésies toxiques. Bulletin Méd.*, 1888.

(25) *Loc. cit.*

(26) *De l'hystérie mercurielle. Soc. Méd. des Hôp. de Paris*, Aug. 12th, 1887.

(27) *Hyst. dans l'intoxication par le sulfure de carbone. Soc. Méd. des Hôp.*, Nov. 9th, 1888.

(28) *Nouvelles recherches sur l'intoxication spéciale que détermine le sulfure de carbone. Acad. de Méd.*, 1863.

(29) Michaux. *Thèse de Paris*, 1886.

(30) *Loc. cit.*

(31) *De l'apoplexie hystérique. Bull. Soc. Méd. des Hôp. de Paris*, Aug. 13, 1886; see also Achard *Apoplexie hystérique*, Th. de Paris, 1887; *Letulle. De l'hystérie saturnine, Semaine médicale*, 1887, and *loc. cit.* Dreyfous *De l'hystérie alcoolique, Union Méd.*, November and December, 1887.

(32) *Ueber Encephalopathia saturnina. Archiv. Psych. and Nervheilk*, 1888, vol. xix., p. 620.

(33) Oppenheim. *Wie sind die Erkrankungen, etc., Berlin Klin. Wochensch.*, 1888, No. 9.

(34) *Leçons sur les mal. du syst. nerv.*, vol. iii., pp. 250 and 268.

(35) *Loc. cit.*

(36) *L'hérédite dans les maladies du syst. nerv., Thèse agrég.* 1886.

(37) *Leçons sur les mal. du syst. nerv.*, Vol. iii. App.

(38) *Soc. de Biol.*, November, 1886.

(39) *Leçons sur deux cas d'Hystérie*, 1890.
(40) HUCHARD. *Etudes de clinique médicale.*
(41) FAÏD. *Troubles de la sensibilité générale dans la période secondaire de la syphilis*, Thèse de Paris, 1870.
(42) MARMISSE. *Gaz. Méd. de Bordeaux*, 1876; *Ricoux. Gaz. hebd. de Méd. et de chir.*, 1878.
(43) GILLES DE LA TOURETTE, BLOCQ ET HUET, *Cinq cas de mal. de Friedreich, Nouv. Icon. de la Salpétrière*, 1888.
(44) GILLES DE LA TOURETTE, *L'hysterie dans l'Armée Allemande. Nouv. Icon. de la Salpêtrière*, 1889; ANDRÉE AND KNOBLAUCH, *Ueber ein Fall von Hystero-Epilepsie bei einem Mann, Berlin-Klin. Woch.*, Mar. 11, 1890.
(45) *Essai sui l'état mental des Hystériques. Thése de Paris*, 1890.
(46) *Loc. cit.*, p. 117.
(47) For a short account of these *see* M. BLOCQ: "Des Stigmates Hystériques," *Gaz. des Hôp*, Jan. 23, 1892.
sur le corps des hommes, 1611.
(48) *Des marques des sorciers et de la réelle possession que le diable prend.*
(49) *Leçons sur les maladies du système nerveux*, vol. iii., p. 345, 1887.
(50) *Centralblatt für, Nerven. krankheiten*, 1879, No. 9, p. 193.
(51) *Leçons sur les maladies du système nerveux*, vol. i., p. 303.
(52) *Le sang dans l'hystérie normal, Progrès Méd.*, Feb. 14, 1891.
(53) *Recherches sur l'absorption souscutanée dans l'hemianœsthésie hystérique.* Thése de Lyon, 1890.
(54) *Anesthésie et ataxie hysteriques, Archives générales de medécine*, 1864.
(55) *Les anesthésies hystériques des muqueuses et des organes des sens et les zones hystérogènes des muqueuses, Thèse de Bordeaux*, 1887.
(56) *L'hystérie et le larynx. Annales des maladies de l'oreille*, vol. vii., pp. 30-41, 1887.
(57) *Op. cit.*
(58) CHARCOT. *Leçons sur les Maladies du Système Nerveux*, vol. iii., p. 441.
(59) *Études cliniques sur l'hystéro-epilepsie*, 1st Ed., 1881, p. 555. See also DE WATTEVILLE, Critical Digest in BRAIN, vol. i., p. 544.
(60) *Anal. in Canstatt's Jahresber*, 1859, vol. i., p. 189.
(61) *Sur la théorie physique de la métallothérapie. Compt. rend., Soc. de Biologie*, 1879, p. 401.
(62) *Leçons du Mardi*, vol. i., 1887-8. *Leçons* 16 & 28.
(63) *Op. cit.*, p. 295.
(64) *Notes pour servir pour l'histoire de l'hystéro-epilepsie. Archiv. du Neurol.*, 1882, p. 281, *et seq.*
(65) *Medical News*, Philadelphia, January 11th, 1890.
(66) *Anesthésie de la rétine. Communication á l'Académie de Médecine de Bruxelles*, p. 11, *et seq.*
(67) *De l'anesthésie rétinienne. Op. cit.*
(68) *Annales d'oculistique*, May—June, 1878.
(69) PARINAUD. *Op. cit.*
(70) *Leçons du mardi*, 1887-88, p. 344.
(71) *Sur l'existence du retrécissement du champ visuel dans la syringomyélie, Médecine Moderne*, Aug. 28, 1890, p. 685.
(72) *Contributions á l'étude du champ visuel dans la syringomyélie et la maladié de Morvan*, Thèse de Paris, May, 1891.
(73) *L'Epilepsie et les épileptiques.*
(74) *Ueber Vorkommen Bedeutung des sensorischen anœsthesie bei Erkrankungen der central Nevensystem. Archiv. für Psychiatrie*, 1884, vol. xv.
(75) *De l'épilepsie dans ses rapports avec les functions visuelles*, Thèse de Paris, 1885, p. 11.
(76) *Hysterische Anœsthesie.* Prag. Vierteljahrs, 1851, p. 72.
(77) *Affections hystériques des muscles oculaires.* Archiv. d'ophthalm. No. 6, 1886, Nos. 2 and 4, 1887, Ann. d'oculi., Nov., Dec., 1887.
(78) *Archives de Neurol.* May, 1891.
(79) *Op. cit.*

(80) *Brit. Med. Jour.*, II. p. 1248, 1884.
(81) *Ueber Hysterie der Kinder. Archiv. f. Kinderheilk.*, 1884.
(82) *Mydriasis, Dict. Encyclop. des Sciences Med.*
(83) *Bulletin de la Clinique des Quinze-Vingts*, July, September, 1883.
(84) *Revue de Médecine*, pp. 337 & 513, 1888.
(85) *Diseases of the Nervous System*, p. 66, 1888.
(86) *Compte rendu du service ophth. pour l'année* 1888.
(87) *Société Médicale des Hôpitaux*, January 26, 1891.
(88) *Leçons sur les Maladies du syst. nerv.*, vol. iii., p. 477.
(89) *Archiv. de Neurol.* Nos. 34, 35, 1890.
(90) *Thèse de Paris*, 1877.
(91) *Op. cit.*, vol. iii., p. 387.
(92) SOLLIER. *Hystérie infantile à forme convulsive.* France Médicale, Jan. 2, 1892.
(93) *Etude sur la méningite tuberculeuse de la adulte.* Thèse de Paris, 1884.
(94) *Bulletins de la Soc. Méd. des Hôpitaux.* May 28, 1891, p. 258.
(95) *Nouvelle Iconographie de la Salpêtrière*, vol. i., 1888, p. 13.
(96) BABINSKI. *De le migraine ophthalmique hysterique. Archiv. de Neurol.*, November, 1890, p, 305.
(97) See *Progrès Médical*, Dec. 22, 1883, p. 1034.
(98) *Progrès Médical*, Oct. 9, 1886.
(99) *Société de Biologie*, 1876.
(100) *Riforma medica* 1885, and *Semaine Médicale*, 1886.
(101) *Archives de Neurologie*, 1885.
(102) *Revue de Médecine*, June, 1887.
(103) *Lo Sperimentale*, Janvier, 1887.
(104) *Medicina Contemporanea*, 1883, No. 1, p. 6.
(105) *Archives de Neurol.*, 1888, vol. xv. See BRAIN, 1891, p. 557.
(106) *Policlinique de Mardi*, March 5, 1889.
(107) See Charcot, *Policlinique de Mardi*, January 24, 1888.
(108) *Leçons sur les maladies du Système Nerveux*, vol. iii. *Leçons du Mardi*, vol. i., p. 398; also *Des Tremblements hystériques, Progrès Méd.* Sept. 6, 13, 1890.
(109) *Des Tremblements hystériques, Progrès Med.* Sept. 14, 21, 1889.
(110) *Contribution à l'etude clinique des tremblements hystériques.* Paris, 1891.
(111) *On the simulation of Hysteria by Organic Diseases of the Nervous System.* London, 1891.
(112) *Contributions a l'étude des syndromes hystériques simulateurs des maladies organiques de la moelle épénière.* Thèse de Paris, 1891.
(113) *Essai sur la Nutrition dans l'Hystérie.*
(114) *Des troubles de la vue chez les hystériques. Progrès Méd.*, No. 3, 1878.
(115) *Société de Biologie*, November 20, 1886.
(116) See *Leçons sur les Maladies du système Nerveux*, vol. iii., p. 441.
(117) *De l'action suggestive des milieux pénitentiaries sur les détenus hystériques.* Revue de l'hypnotism, November, 1889, p. 131.
(118) *Loc. cit.*, vol. iii., p. 255.
(119) The above account of the Hysterical Mental State is taken from M. Gilles de la Tourette's work.
(120) TAGUET. *Du suicide dans l'Hystérie. Annales Médico-psychologiques.* May, 1877. LEGRAND DU SAULLE. *Les Hystériques*, Paris, 1883. HUCHARD, *Traité des nevroses*, Paris, 1883.
(121) See Critical Digest in BRAIN, 1891, p. 538.
(122) *De l'élimination des Phosphates dans les Maladies du Système Nerveux*, Paris, 1892.